UNIVERSITY OF ALBERTA

EFFECTS OF RESISTANCE TRAINING ON LEFT VENTRICULAR MORPHOLOGY

By

Mark Haykowsky

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Doctor of Philosophy

Faculty of Physical Education & Recreation

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The undersigned certify that they have read and recommend to the Faculty of Graduate studies and Research for acceptance, a thesis entitled **EFFECTS OF RESISTANCE TRAINING ON LEFT VENTRICULAR MORPHOLOGY** submitted by Mark J. Haykowsky in partial fulfillment of the requirements for the degree of **DOCTOR OF PHILOSOPHY.**

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Abstract

The purpose of the series of studies in this thesis was to assess the acute effects of resistance exercise on left ventricular (LV) wall stress and systolic function. In addition, a secondary purpose was to assess the chronic effects of resistance training on LV wall thickness, dimensions and mass in younger, middle-aged and older males.

In chapter III, the acute effects of leg-press exercise performed with a brief Valsalva maneuver on LV wall stress and LV systolic function were assessed. The results revealed that heavy resistance exercise was not associated with changes in LV wall stress or LV systolic function. The attenuated LV wall stress was likely secondary to acute heart-lung interactions and intrinsic alterations in LV geometry that reduced the tension per cross-sectional myocardial area.

The effects of long-term (mean ± S.D.: 10 ± 5 years) resistance training on left ventricular dimensions and mass was assessed in elite male resistance trained athletes (mean age: 33.4 ± 5.9 years) (Chapter IV). The results revealed that long-term resistance training was not associated with alterations in left ventricular wall thickness, diastolic cavity size or estimated left ventricular mass. In addition, no athlete was found to have a mean LV wall thickness above clinically acceptable limits.

The effects of short-term (4.4 ± 3.4 years) versus long-term (18.3 ± 6.6 years) resistance training as performed by elite junior (mean age: 21.1 ± 1.2 years) and master (mean age: 46.0 ± 5.5 years) athletes on left ventricular morphology was assessed (Chapter V). The results revealed that short or long-

term resistance training was not associated with alterations in left ventricular dimensions or mass.

The effects of 16 weeks of resistance training on left ventricular morphology and systolic or diastolic function was assessed in healthy older males (mean age: 68.0 ± 3.4 years)(Chapter VI). The results revealed that four months of resistance training was associated with an increase in upper and lower extremity maximal muscular strength. However, this form of exercise was not associated with changes in left ventricular dimensions, mass, systolic or diastolic function.

The results of these investigations suggest that resistance training was not associated with an acute alteration in LV wall stress. Moreover, 0.3 to 18 years of resistance training was not associated with changes in LV wall thickness, diastolic cavity size or mass in younger, middle-aged or older males. These findings suggest that resistance training may be a sufficient stimulus to increase maximal muscular strength, but is an insufficient stimulus to alter left ventricular morphology.

Dedication

To Michael Haykowsky: Father, Friend and Mentor

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Chapter 1

Introduction

Introduction

Resistance training (RT) has gained popularity as an effective exercise intervention to improve overall physical fitness as well as an adjunct to enhance sport performance (Fleck and Kraemer, 1997). In addition, RT has recently gained acceptance as a safe form of exercise for frail elderly or cardiac patients to increase their maximal muscular strength and functional capacity (Fiatarone and Evans, 1993; Verill and Ribsil, 1996). Despite the benefits of RT to improve skeletal muscle morphology and function (Frontera, et al., 1988; Fiatarone, et al., 1990), this form of training has been reported to be associated with an extreme and potentially harmful increase in arterial pressure which may alter cerebro-vascular or left ventricular (LV) morphology (Haykowsky, et al., 1996; Fagard, 1996).

Previous studies have demonstrated that sub-maximal or maximal resistance exercise was associated with a transient abrupt increase in arterial pressure (MacDougall, et al., 1985; 1992; Lentini, et al., 1993), intra-thoracic pressure (Del Torre, et al., 1990; MacDougall, et al., 1992; Lentini, et al., 1993) and intra-abdominal pressure (Harmon, et al., 1988; Del Torre, et al., 1990). MacDougall and associates (1985) have reported that healthy younger individuals were capable of generating arterial pressures as high as 343/235mmHg during sub-maximal repetitive resistance exercise. Moreover, extreme elevations in arterial pressure (i.e., 480/350mmHg) have been measured during leg-press exercise (MacDougall, et al., 1985). The mechanisms responsible for the RT-mediated pressure load were mechanical

compression of the peripheral vessels by the contracting muscles and increased intra-thoracic pressure associated with performing a brief Valsalva maneuver (MacDougall, et al., 1985). A consequence of chronic pressure overload is that it may be a potent stimulus for left ventricular (LV) hypertrophy.

A series of echocardiographic investigations have demonstrated that RT was associated with alterations in LV morphology mediated by increases in posterior wall thickness, ventricular septal wall thickness, relative wall thickness (ratio of wall thickness to diastolic cavity diameter) and estimated LV mass (Colan, et al., 1987; Sagiv, et al., 1992; Pelliccia, et al., 1993; Abinader, et al., 1996). The mechanism responsible for the RT mediated alteration in LV morphology appears to be secondary to the increased pressure load associated with this form of exercise (Colan, et al., 1992). For example, acute increases in systolic pressure are believed to be associated with a concomitant increase in LV wall stress that can only be minimized by an increase in LV wall thickness (Colan, et al., 1997). Therefore, the RT mediated alteration in LV wall thickness appears to be a physiological adaptation that normalizes LV wall stress (Pelliccia, et al. 1993).

Several studies have revealed that the magnitude of the alteration in LV morphology was related to the age and training experience of the athletes (Nishimura, et al., 1980; Miki, et al., 1994). For example, master athletes (40 to 49 years) with 26.5 years of training experience were found to have a larger diastolic cavity size, posterior wall thickness, ventricular septal wall thickness and estimated LV mass compared to "sport matched" younger (20 to 39 years)

athletes with 4 to 15 years of training experience (Nishimura, et al., 1980). Currently, there has been a paucity of studies assessing the effects of RT in master athletes. However, the findings of Nishimura et al., (1980) suggest that long-term training may result in progressive increases in LV dimensions and mass in middle-aged or older competitive athletes.

A series of echocardiographic investigations have demonstrated that increased age was associated with alterations in LV morphology that were similar to that found secondary to RT (i.e., increased posterior wall thickness, ventricular septal wall thickness, relative wall thickness and estimated LV mass)(Sjorgen, 1971; Gerstenblith, et al., 1977; Lindros, et al., 1994; Gardin, et al., 1995). The mechanism(s) responsible for the age-related alteration in LV morphology are unclear. However, these changes may be secondary to the increased arterial stiffness and arterial pressure associated with senescence (Lewis and Maron, 1992; Lakatta, 1993).

Currently, RT has gained widespread acceptance as a safe and effective exercise intervention for older adults to offset or reverse the age-mediated decline in muscle mass and strength (Frontera, et al., 1988; Fiatarone, et al., 1990). However, a series of studies have demonstrated that older adults are capable of generating relatively high systolic pressures (i.e. 247mmHg to 261mmHg) when performing sub-maximal resistance exercise (McCartney, et al.,1993; Benn, et al., 1996). Therefore, it is possible that the age-related increase in pressure load together with the RT-mediated pressure load may combine to produce an even greater stimulus for alterations in LV morphology.

Statement of the problems

At the present time there are certain controversies and limitations surrounding the studies that have assessed the acute cardiovascular responses during resistance exercise or the chronic effects of RT on LV morphology.

The first controversy that requires further clarification is the acute effects of RT on LV wall stress. As previously discussed, a widely held belief in sport cardiology has been that the increased pressure load during resistance exercise was associated with a concomitant increase in LV wall stress. However, from the perspective of cardiac mechanics this relationship may not hold true. Since LV wall stress is defined as the product of LV transmural pressure (i.,e., intra-vascular pressure minus intra-thoracic pressure) and LV geometry, acute changes in either variable may alter LV wall stress. For example, Lentini et al., (1993) have shown that the increased systolic pressure during resistance exercise was secondary to the heightened intra-thoracic pressure associated with performing a brief Valsalva maneuver. However, the pressure to which the heart was "exposed" (i.e., LV transmural pressure) was lower than that predicted by the intra-vascular pressure alone. Therefore, a brief (~ 2 to 3 second) Valsalva maneuver during resistance exercise may reduce the pressure to which the heart is "exposed" and as a consequence may minimize the elevation in LV wall stress. In addition, Galanti and associates (1992) have recently demonstrated that the heart alters its geometrical configuration during acute isometric exercise in such a manner that the tension per cardiac fiber was reduced and as a consequence LV wall stress was unaltered. Based on the

findings of Galanti et al., (1992) and Lentini, et al., (1993) it may be possible that acute heart-lung interactions and intrinsic changes in LV geometry may occur during resistance exercise to reduce LV wall stress. Therefore, further studies are required to assess the acute LV wall stress response during resistance exercise.

A limitation of the echocardiographic studies assessing the effects of RT on LV dimensions and mass was that the subjects were younger (< 25 years) individuals who had been training for a relatively short period of their life (< 5 years). Based on this limitation two questions require further study. First, the effects of long-term (> 10 years) RT and the associated pressure load on LV dimensions and mass remain unknown. In addition, the upper limits of LV wall thickening with long-term RT require further study. Second, the short-term versus long-term effects of RT on LV dimensions and mass remain unanswered.

Lastly, RT has recently gained acceptance as a safe and effective exercise intervention for healthy older adults. However, before RT can be classified as "safe" for older adults further studies are required to ensure that the this form of training does not result in a harmful deterioration in the size and systolic or diastolic function of the heart.

Significance of the studies

The findings of this dissertation will help clarify the acute effects of RT on LV wall stress as well as the chronic effects of RT on LV dimensions and mass. These findings will also determine if there are potential protective mechanisms (i.e., a brief Valsalva maneuver) that may be performed during RT to minimize

LV wall stress. In addition, the upper limits of LV wall thickening with long-term RT will be determined which will help sport cardiologists differentiate between physiologic hypertrophy secondary to RT (i.e. the "athletes heart") and pathologic abnormalities associated with pressure overload hypertrophy. Lastly, the findings of the study assessing the effects of RT on LV morphology and systolic or diastolic function in older adults will determine if this form of exercise can be classified as "safe" for this population.

Study Objectives

The objectives of this dissertation are to assess:

1) The acute effects of resistance exercise on LV end-systolic wall stress and LV systolic function.
2) The effects of long-term (> 10 years) RT on LV dimensions and mass.
3) The effects of short (< 5 years) versus long-term RT on LV dimensions and mass.
4) The effects of 16 weeks of RT on LV morphology, systolic and diastolic function in healthy older males.

Hypothesis

1) Resistance exercise will not be associated with an acute alteration in LV end-systolic wall stress or systolic function.
2) Long-term RT will not be associated with alterations in LV wall thickness, cavity size and estimated LV mass.
3) Short or long-term RT will not be associated with alterations in LV wall thickness, cavity size and estimated LV mass.

4) Sixteen weeks of RT will be not be associated with an alteration in LV dimensions, mass or systolic and diastolic function in healthy older males.

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Chapter II

Review of Literature

In this brief review, an examination of the relevant literature regarding the acute hemodynamic responses associated with resistance training (RT) as well as the chronic effects of RT on left ventricular (LV) morphology will be reported. In addition, the effects of aging on LV morphology and systolic or diastolic function will be explored. Lastly, the role of RT to offset the age-mediated decline in muscle mass and strength will be discussed.

In this review, resistance training (synonymous with strength-training or weight-training) is defined as "a type of exercise that requires the body's musculature to move (or attempt to move) against an opposing force, usually presented by some type of equipment" (Fleck and Kraemer, 1997). Moreover, athletes who participate in bodybuilding, olympic-weightlifting and powerlifting will be referred to as resistance trained athletes since they all perform resistance exercises to increase maximal muscular strength and/or muscle mass.

Acute hemodynamic responses during resistance training

Upper or lower extremity resistance exercise has been shown to be associated with a transient abrupt increase in heart rate, systolic, diastolic and mean arterial pressure (MacDougall, et al.,1985,1992,1994; Fleck, and Dean,1987; Palatini, et al.,1987; Del Torre,1990; Lentini, et al.,1993; Narloch,1995). Healthy younger individuals have been able to generate arterial pressures as high as 343/235mmHg during sub-maximal repetitive resistance exercise (Table 2.1)(MacDougall, et al., 1992). Moreover, extreme elevations in arterial pressure (e.g.480/350mmHg) have been reported during leg-press

Table 2.1 Acute blood pressure response during sub-maximal and maximal resistance exercise

Study	Age (Years)	Training Status	Exercise	Intensity (% 1 RM)	SBP (mmHg)	DBP (mmHg)	MAP (mmHg)
Del Torre (1990)	26.6±6.3	TDBB	SAC SQT	60-100 60-100	223 261	156 192	178 215
Fleck and Dean (1987)*	23.0±1.1 23.3±1.1	UT NOV	KE	50 70	160-181 158-190	125-136 116-150	106 128
Lentini (1993)	23.0±1.5	TDWL	LP	95	270	183	212
MacDougall (1985)*	22-28	TDBB	SAC SLP	95 95	231.9 261.6 480	173.2 194.2 350	193 217 393**
MacDougall (1992)	21-32	NOV-TDBB	LP	85	343	235	271
Narloch (1995)	25-35	TDBB, CY,RN	LPNVM LPVM LPNVM LPVM	85% 85% 100 100	178 267 198 311	156 239 175 284	163 248 206 293

(1 RM, one-repetition maximum; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; TDBB; trained bodybuilders; UT; untrained; NOV, novice; TDWL, trained weightlifter; CY, cyclists; RN, runners; SAC; single arm curl; SQT, squat; KE, knee extensions; LP, leg press; SLP, single leg press; LPNVM, leg-press performed without a Valsalva maneuver; LPVM, leg-press performed with a Valsalva maneuver; *, visually estimated from published data; **, highest pressure recorded in one individual).

exercise (MacDougal, et al., 1985). The underlying mechanism(s) responsible for the heightened blood pressure response are the combination of 1) the pressor response; 2) increased mechanical compression of vascular vessels by the contracting musculature and 3) positive swings in intra-thoracic pressure associated with performing a brief (~ 2 to 3 second) Valsalva maneuver (MacDougall, 1985,1992,1994).

Acute blood pressure response during resistance training: Role of the central "feed-forward" and peripheral "feedback" responses

The characteristic hemodynamic responses during RT have been shown to be mediated, in part, by two neural mechanisms which innervate the cardiovascular control center in the medulla (Mitchell, 1990). The central or "feed-forward" mechanism originates in a central portion of the brain and activates, in a parallel fashion, the motor cortex and the cardiovascular control center. The peripheral "feedback" mechanism originates in the skeletal muscles and stimulates the cardiac control center via group III and IV muscle afferents (Mitchell, 1990). The result of stimulating the cardiovascular control center was a reflexive increase in sympathetic output with a concomitant decrease in parasympathetic nervous system activity resulting in the characteristic pressor response (i.e. increased heart rate, systolic, diastolic and mean arterial blood pressures) (Mitchell, 1990).

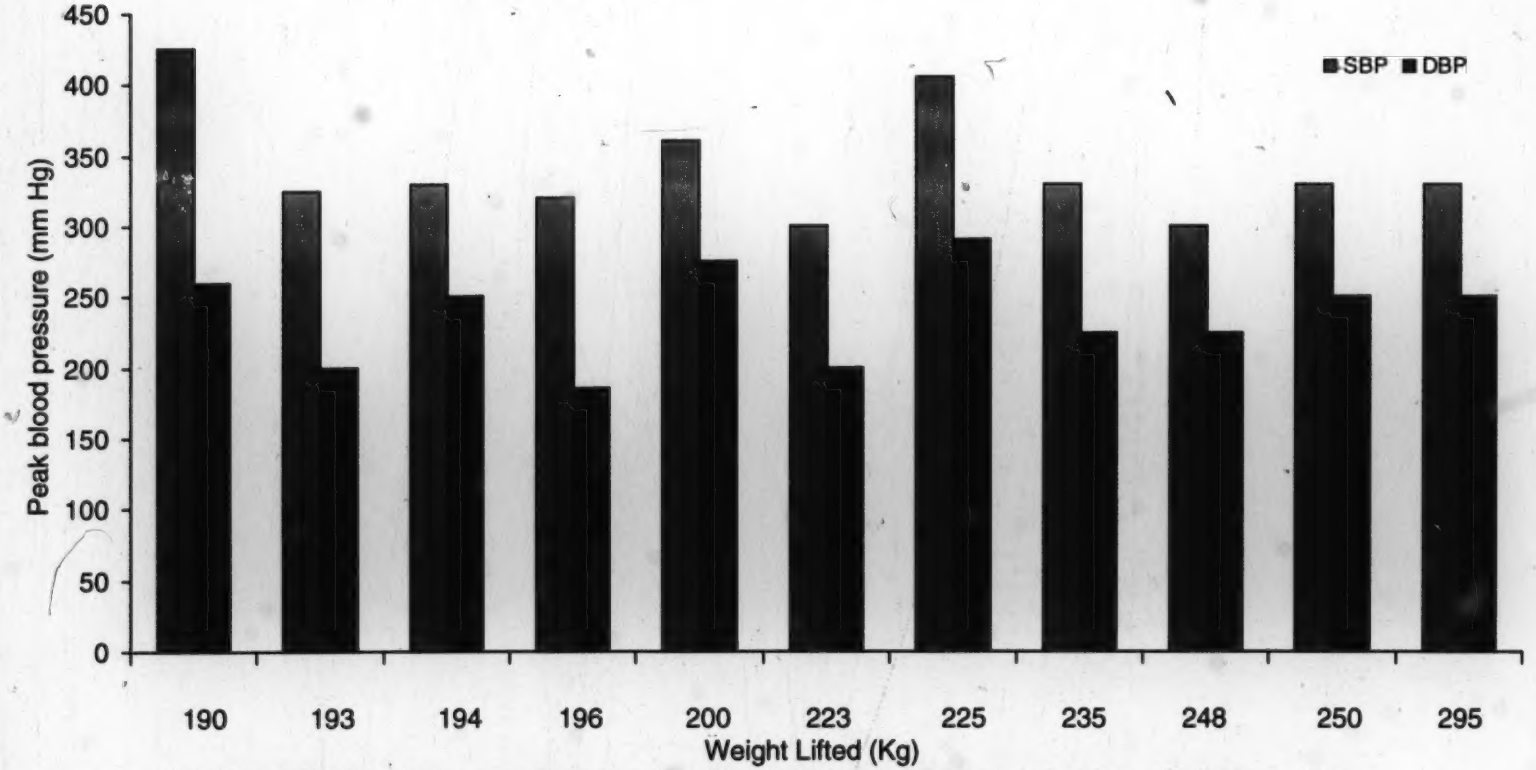
Effects of muscle strength and muscle mass on the resistance mediated blood pressure response

The magnitude of the blood pressure response during RT appears to be independent of the absolute weight lifted or muscle mass (MacDougall et al.,1992). Figures 2.1 and 2.2 show that individuals with reduced muscle mass and strength were capable of generating extreme elevations in arterial blood pressure when performing bilateral leg-press exercise (MacDougall, et al., 1992). Moreover, the greatest elevation in arterial pressure (i.e. 425/250 mm Hg) was generated by the individual with the lowest absolute muscle strength and third lowest bilateral quadriceps cross-sectional area (MacDougall, et al., 1992). Accordingly, the magnitude of the RT mediated alteration in arterial pressure appears to be related to the degree of voluntary effort (central mediated feed-forward neural mechanisms) rather than the absolute weight lifted or size of the contracting skeletal musculature (MacDougal, et al.,1992,1994).

Effect of a brief Valsalva maneuver on the systolic pressure during resistance exercise

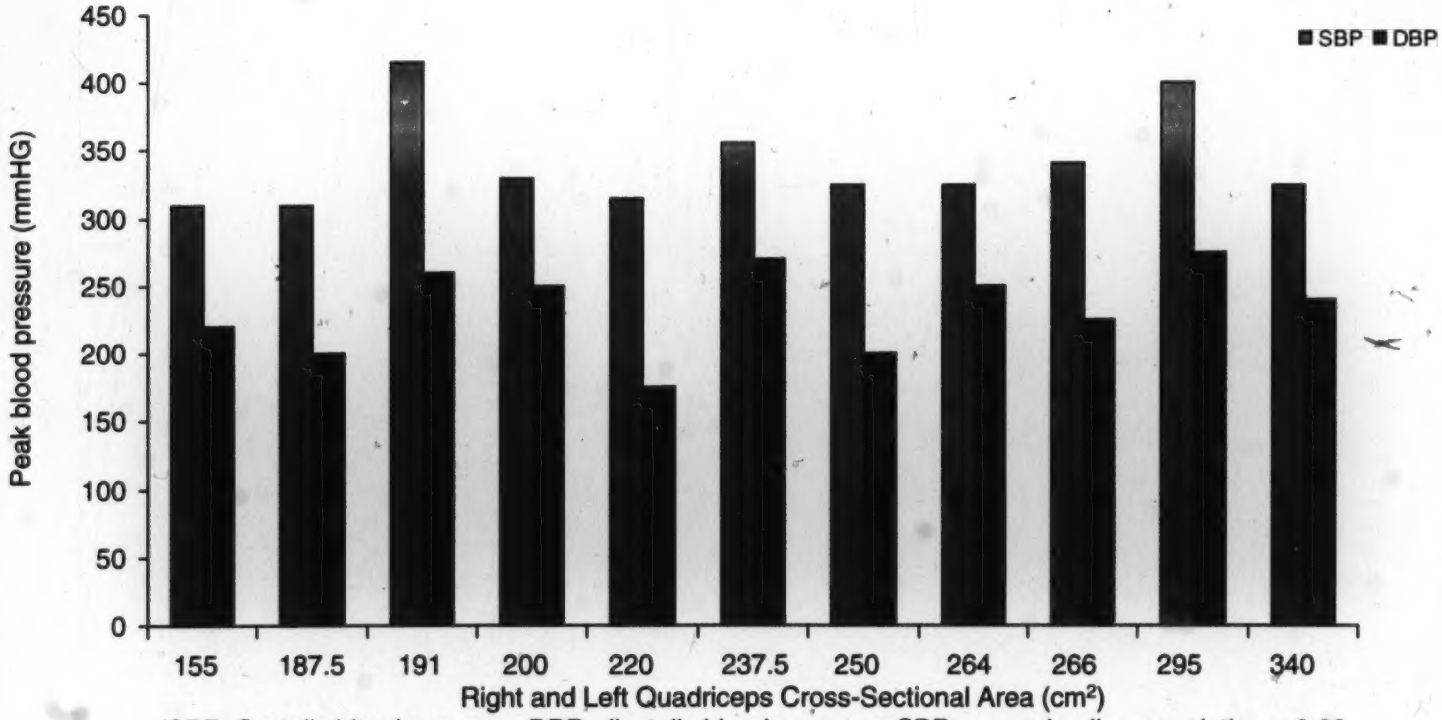
The heightened blood pressure response associated with RT has been attributed, in part, to the increase in intra-thoracic pressure (ITP) associated with performing a brief (~2 to 3 second) Valsalva maneuver (MacDougal, et al.,1985, 1992; Palatini, et al., 1987; Lentini, et al.,1993; MacDougall,1994). The magnitude of the increase in intra-thoracic pressure during RT has been shown to be similar to that which occurs at 70% of a maximal voluntary Valsalva

Figure 2.1 Peak systolic and diastolic blood pressure versus absolute weight lifted



(SBP, systolic blood pressure; DBP, diastolic blood pressure; SBP regression line correlation = -0.33 non significant; DBP regression line correlation = 0.12 non significant; data estimated from published figure from MacDougall et al., 1992).

Figure 2.2 Peak systolic and diastolic blood pressure versus quadriceps muscle size



(SBP, Systolic blood pressure; DBP, diastolic blood pressure; SBP regression line correlation = 0.22 non-significant; DBP regression line correlation = 0.38 non-significant; data estimated from published figure from MacDougal et al., 1992)

maneuver without lifting (~100 mm Hg; MacDougall, et al.,1992;1994). However, extreme elevations in intra-thoracic pressure (i.e. 256-261 mmHg) have been recorded during resistance exercise (Compton and McNHill, 1973).

Since the heart and lungs are intimately related within the cardiac fossa (Butler, 1983) changes in intra-thoracic pressure can directly affect systolic pressure. For example, Hamilton and Buda and their co-workers (1944, 1979) have shown that the heightened intra-thoracic pressure associated with a brief Valsalva maneuver (without lifting) was transmitted directly to the arterial vasculature resulting in an increase in systolic pressure. However, the pressure to which the heart was "exposed" (i.e., LV transmural pressure) was not elevated above resting values. Therefore, positive swings in intra-thoracic pressure directly elevate systolic pressure, however, the increased intra-thoracic pressure also acts as a safety factor by "preventing any rise in vascular transmural pressure which could cause vascular damage in the large vital vessels in either the thorax, cerebro-spinal canal or the abdomen" (Hamilton, 1962). Based on the above heart-lung interactions, it has recently been suggested that a brief Valsalva maneuver should not be discouraged during RT (MacDougall et al 1992; Lentini, et al. 1993; Haykowsky, et al., 1996).

Effects of resistance exercise on intra-muscular pressure

A series of investigations have demonstrated that intra-muscular pressure may reach values as high as 570mmHg-1676mmHg during static muscular contractions (Sylvist, et al.,1959; Sejersted and Hargens,1995; Ameredes, et al.,1997). The magnitude of the alteration in intra-muscular pressure was related

to the amount of force generated, but, was independent of the mode or speed of muscle contraction (Sejersted and Hargens, 1995). The consequence of the extreme intra-muscular pressure during muscular contractions is the occlusion of peripheral muscle blood flow resulting in an increase in peripheral vascular resistance and arterial pressure.

Effects of resistance training on cardiac volumes, output and contractility

The acute effects of the heightened pressure load on cardiac volumes, output and LV systolic function have not been well studied. However, Lentini and associates (1993), using 2-dimensional transthoracic echocardiography, have recently demonstrated that sub-maximal bilateral leg-press exercise was associated with a decline in LV end-systolic volume, end-diastolic volume and stroke volume. The attenuated preload was offset by increases in heart rate and LV contractility resulting in an elevation in cardiac output and ejection fraction. The mechanism(s) responsible for the attenuated cardiac volume response appear to be due, in part, to the heightened pressure load and increased vascular resistance associated with this form of exercise.

In summary, sub-maximal or maximal resistance exercise has been shown to be associated with hemodynamic alterations, including increases in heart rate, systolic pressure, diastolic pressure, mean arterial blood pressure, intra-thoracic pressure, intra-muscular pressure and systemic vascular resistance resulting in a concomitant decline in end-diastolic volume, end-systolic volume and stroke volume. The underlying physiological mechanisms eliciting these responses are due, in part, to the heightened intra-thoracic and

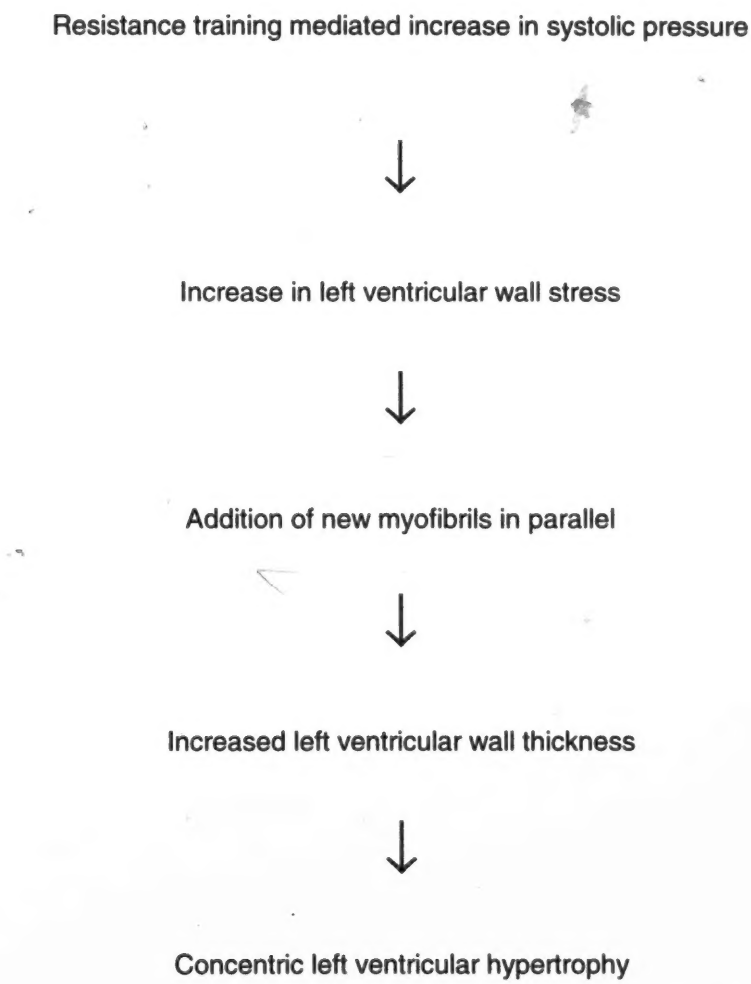
intra-muscular pressure secondary to contraction of the respiratory and skeletal muscles, respectively. The consequence of the heightened pressure load is that it may be a potent stimulus to alter the size of the heart (Colan, 1992).

Effects of resistance training on left ventricular morphology

Resistance training has been shown to be associated with alterations in LV morphology including increases in posterior wall thickness, ventricular septal wall thickness, relative wall thickness (ratio of wall thickness to cavity dimension) and estimated LV mass (Kanakis and Hickson, 1980; Agati, et al., 1985; Lusiani, et al., 1986; Colan, et al., 1987; Deligiannis et al., 1988; Peliccia, et al., 1993; Abinader, et al., 1996; Fagard, 1996). The mechanism(s) responsible for RT mediated LV hypertrophy remain uncertain, however, Linzbach (1960) has suggested that a major stimulus controlling the size of the LV was the systolic tension or force imposed on the myocardial fibers. Grossman et al., (1975) postulated that sudden increases in systolic pressure (without lifting) resulted in a concomitant elevation in LV wall stress which (in accordance to the Laplace law) could only be normalized by an increase in LV wall thickness (Figure 2.3). Based on the above paradigm, a widely held belief in sport cardiology has been that the RT mediated pressure load and wall stress may be a potent stimulus to increase LV wall thickness and mass (Colan, 1992).

Previous echocardiographic studies assessing the "athletes heart" have found that the magnitude of the alteration in LV morphology was related to the length of training experience as master athletes had larger LV dimensions and mass compared to sport-matched younger athletes (Nishimura, et al., 1980; Miki,

Figure 2.3 Load induced cardiac hypertrophy


Resistance training mediated increase in systolic pressure
Increase in left ventricular wall stress
Addition of new myofibrils in parallel
Increased left ventricular wall thickness
Concentric left ventricular hypertrophy

(concentric hypertrophy occurs when the left ventricular septal or posterior wall increase in size without a concomitant alteration in diastolic cavity dimension, Adapted from Grossman, et al., 1975).

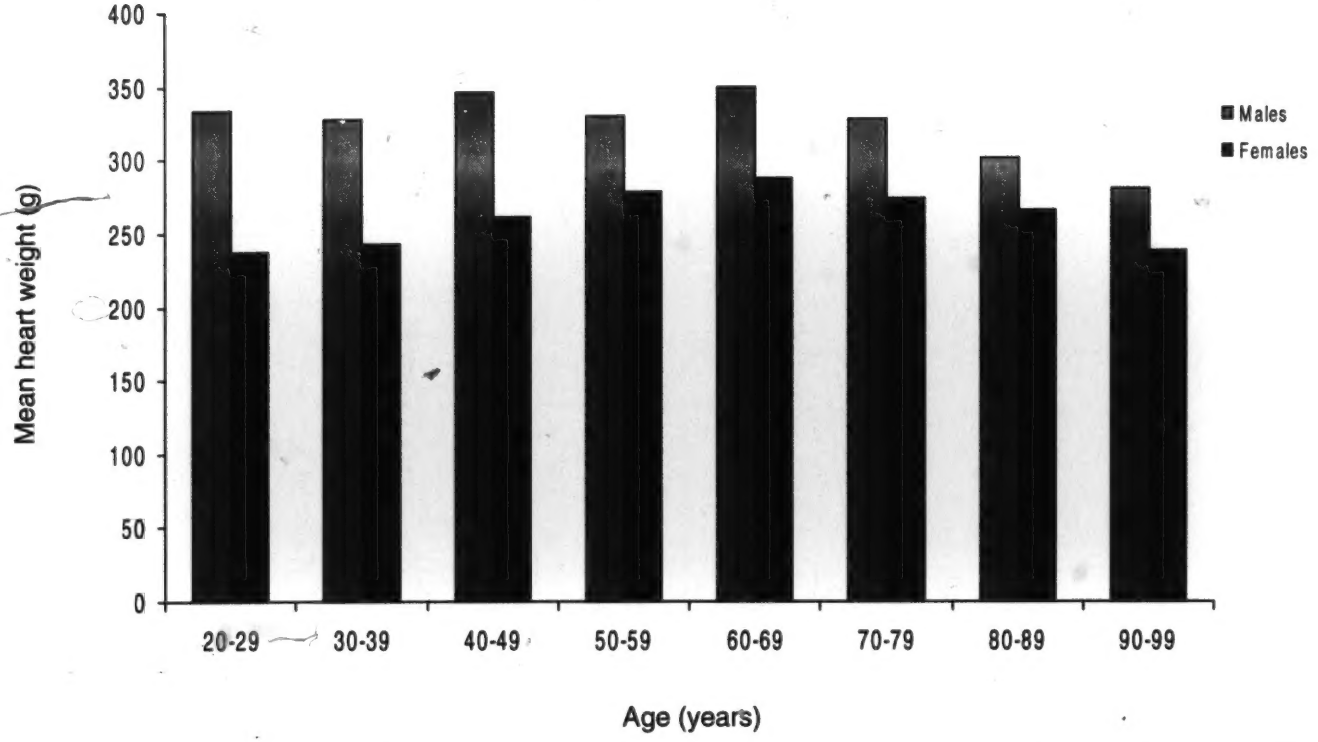
et al., 1994). Currently, a limitation of the studies assessing the effects of RT on LV morphology has been that the subjects were younger individuals (< 25 years) who had been training for a relatively short period of their life (< 5 years). Therefore, the effects of long-term RT (> 10 years) and the subsequent pressure load on LV dimensions and mass are not well known. In addition, increased age has been associated with alterations in LV morphology similar to that found secondary to RT (i.e., increased LV wall thickness and estimated LV mass [Lewis and Maron, 1992]). Therefore, it may be possible that aging combined with RT may be a potent stimulus to alter LV dimensions and mass.

The second part of this review will assess the effects of aging on LV morphology and systolic or diastolic function. In addition, the role of RT to offset the age-mediated decline in muscle mass and strength will be examined.

Age-related alterations in left ventricular dimensions and mass

Increased age has been shown to be associated with alterations in left ventricular (LV) morphology including increases in posterior wall thickness, ventricular septal wall thickness, relative wall thickness and estimated LV mass (Sjorgen, 1971; Gerstenblith, et al.,1977; Lewis and Maron,1992; Lindroos, et al.,1994; Gardin, et al.,1995). Kitzman and associates (1988) assessed the time course of the alteration in LV morphology and found that mean heart weight progressively increased from the third to seventh decades of life followed by a steady decline thereafter (Figure 2.4). The senescent mediated increase in LV mass was secondary to cardiomyocyte hypertrophy as the number of cardiac

Figure 2.4 Effects of aging on mean heart weight



(Adapted from Kitzman, et al., 1988)

muscle fibers has been shown to decline with age (Olivetti, et al, 1991). The underlying mechanism(s) responsible for the alteration in LV morphology have not been fully studied. However, these changes may be secondary to underlying coronary artery disease, hypertension or obesity as healthy individuals without the above risk factors did not have alterations in LV mass with increasing age (Dannenberg, et al., 1989; Figure 2.5).

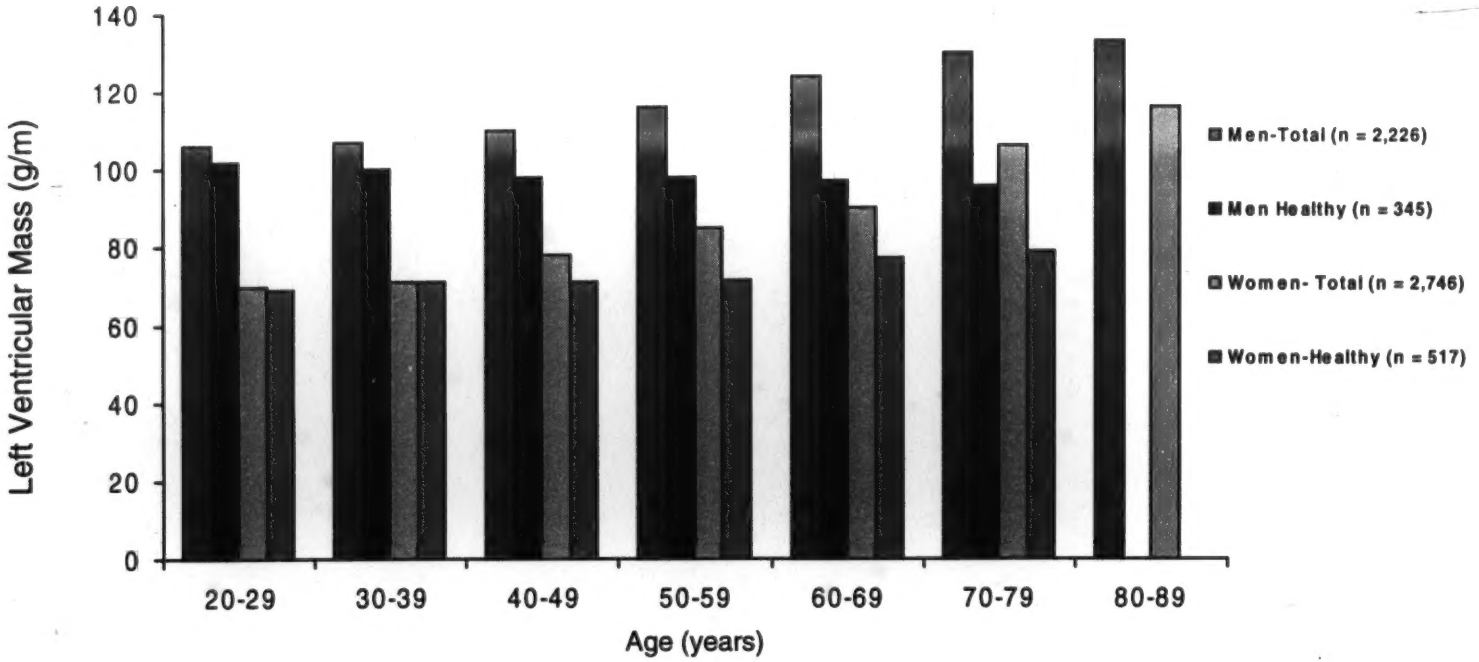
Effects of aging on left ventricular systolic function

A series of studies have demonstrated that aging was not associated with changes in resting LV systolic function in healthy individuals without underlying coronary artery disease (Lakatta, 1993; Fleg, et al., 1995a). Rather, the mechanisms that maintain LV systolic function in younger individuals are altered with age. For example, at rest healthy older individuals rely on an increased end diastolic volume (and the Frank-Starling mechanism) to maintain stroke volume, ejection fraction and cardiac output. During peak aerobic stress, older individuals continue to rely on the Frank-Starling mechanism to maintain stroke volume, however, the decline in peripheral vascular conductance coupled with a blunted heart rate and LV contractile response result in an attenuated ejection fraction and cardiac output (Table 2.2)(Fleg, et al., 1995a).

Effects of aging on left ventricular diastolic function

Increased age has also been associated with an alteration in resting and exercise diastolic function (Lewis and Maron, 1992; Schulman, et al.,1992; Fleg,1995b). Previous studies, using pulsed-Doppler echocardiography, have

Figure 2.5 Echocardiographic left ventricular mass by age and sex in the Framingham study



Adapted from Dannenberg, et al., 1989

Table 2.2 Age-related alterations in resting and exercise left ventricular systolic function

Variable	Rest	Peak Exercise	Cardiac Reserve (Change from rest to peak exercise)
HR (beats/min)	↓	↓	↓
SBP (mmHg)	↑	↔	
MAP (mmHg)	↑	↑	
ESVI (ml)	↑	↑	↑
SVI (ml/beat)	↑	↔	↔
EF (%)	↔	↓	↓
SBP/ESVI ($mmHg/ml/m^2$)	↔	↓	

(HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; ESVI; end-systolic volume index; EF, ejection fraction; SBP/ESVI, an indirect measure of left ventricular contractility; ↓, significant age-related decline; ↑, significant age-related increase; ↔, no significant age effect; Adapted from Lakatta, 1993 and Fleg, et al., 1995a)

shown that senescence was associated with a decline in peak early (E) diastolic flow velocity associated with compensatory increase in peak late (or atrial, [A]) flow velocity culminating in an attenuated E/A ratio (Spirito and Maron, 1988; Klein, et al., 1994). The age-mediated decline in diastolic function does not appear to be related to underlying fitness levels as master athletes have been shown to have diastolic filling patterns similar to that of age-matched sedentary individuals (Fleg, et al.,1995b).

In summary, increased age has been shown to be associated with an alteration in the size and systolic and diastolic function of the heart. At rest, advanced age was associated with a greater utilization of the Frank-Starling mechanism to maintain stroke volume, ejection fraction and cardiac output. Furthermore, the age-mediated reduction in cardiac reserve during peak aerobic stress may result in a concomitant decline in maximal aerobic power. However, it is also possible that the age-related decrease in muscle mass may play an important role in the attenuated functional capacity (Fleg and Lakatta,1988)

Effects of aging on skeletal morphology

A hallmark of the aging process has been the progressive decline in skeletal muscle mass (Fleg and Lakatta, 1988; Ayogai and Sheperd,1992; Lexell,1988, 1992, 1995) associated with a concomitant increase in non-muscle tissue (Rice, et al., 1989; Overend, 1990). The alteration in skeletal morphology begins half way through the third decade of life and continues until 50% of the muscle volume is lost by the ninth decade. The age-related decline in muscle

mass, coined sarcopenia (Evans, 1995), has been attributed to a loss and/or a reduction in the size of muscle fibers (Lexell, 1988, 1992, 1995).

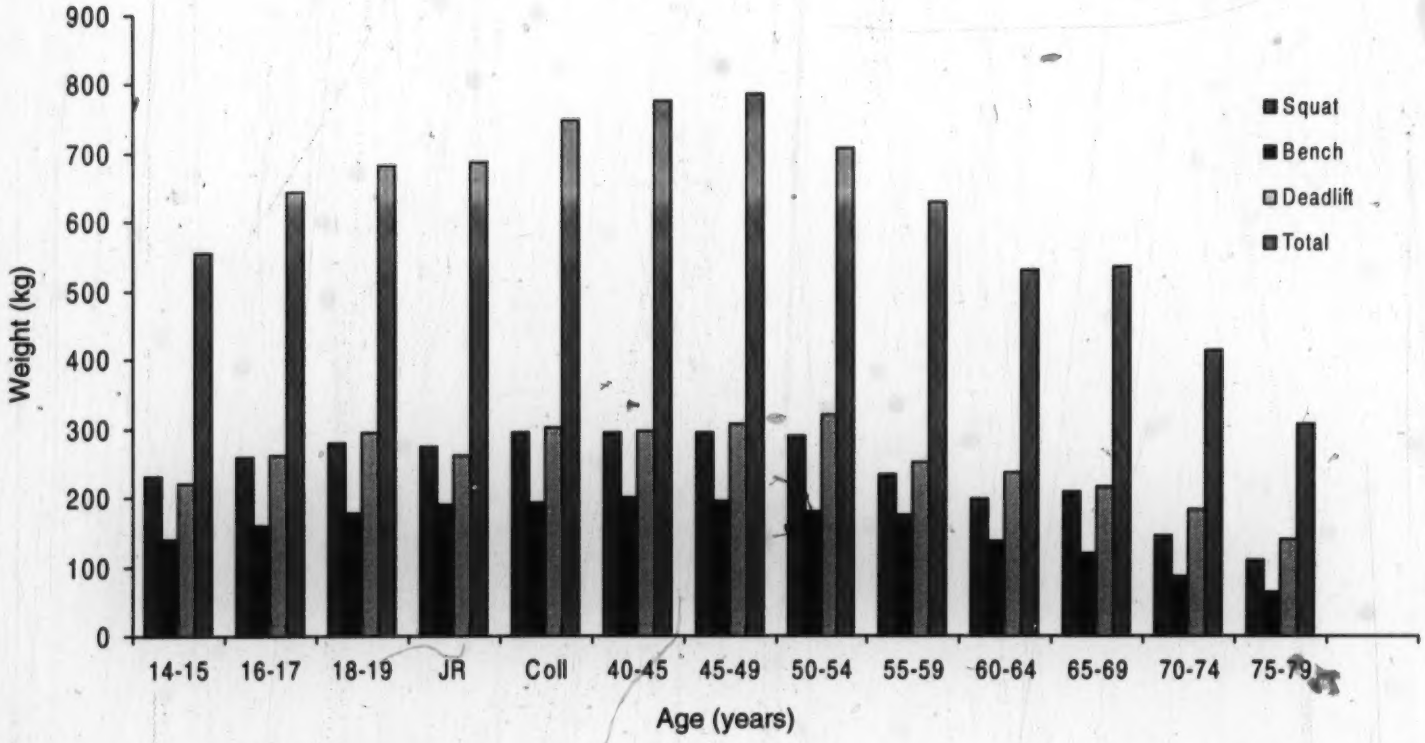
Skeletal muscle fiber size of upper and lower limb muscles remain relatively constant until 50 to 60 years of age followed thereafter by a reduction in the size of the type II muscle fibers (Aniansson,1980; Grimby and Saltin,1983; Lexell,1988, 1992, 1995, Ayogai and Sheperd,1992; Rogers and Evans,1993). The oxidative, type I, muscle fibers appear to be resilient to the aging process as no alteration in their cross sectional area was observed between 20-80 years of age (Lexell, 1992, 1995; Porter, et al., 1995).

The absolute decline in muscle cross sectional area has been shown to be moderate compared to the overall reduction in muscle volume suggesting that a loss of muscle fibers may play a role in the decline in muscle volume (Grimby and Saltin, 1983). Lexell (1992) confirmed that the 18% decline in muscle fiber number between the fourth and eight decades of life was the main determinant of the age-related reduction in muscle volume. Therefore, advanced age has been shown to be associated with alterations in skeletal morphology mediated by a loss and atrophy of the type II fibers with minimal alteration in oxidative fibers (Lexell, 1995).

Effects of aging on maximal muscular strength

A series of cross-sectional studies have revealed that increased age was associated with a decline in maximal muscular strength (Figure 2.6)(Porter, et al., 1995; Lindle, et al., 1997). The onset of the decline in strength begins around the fourth decade of life culminating in a 50% reduction by the ninth decade

Figure 2.6 Effects of aging on maximal muscular strength in elite powerlifters



(JR, < 23 years; Coll, collegiate students; records for athletes competing in the American drug free powerlifting 82.5 kg class)

(Tseng, 1995). The time-course of the decline in muscle strength appears to be accompanied by a parallel reduction in skeletal mass suggesting that the former may be due in part to the latter (Rodgers and Evans, 1993). The causal relationship between the alteration in muscle strength and mass was demonstrated by Frontera and associates (1991) who found that the age-related decline in muscle strength was reduced when expressed relative to fat-free mass or skeletal muscle mass. Therefore, the decline in muscle mass appears to be associated with a parallel reduction in maximal muscular strength (Lexell, 1995; Evans,1995, 1996) which may lead to muscle weakness and decreased functional independence (Fiatarone, et al., 1990). Therefore, interventions that can reverse or offset the decline in muscle mass and strength are essential in maintaining older individuals functional independence.

Effects of resistance training to offset the age-related decline in muscle mass and strength

A recent cross-sectional retrospective study revealed that 68 year-old males who participated in long-term (12-17 years) RT had similar muscle strength and cross-sectional muscle fiber areas as younger (28 years) sedentary individuals (Klittgard, et al., 1990). The magnitude of the alteration in muscle mass and strength appeared to be specific to the mode of athletic training as master endurance trained athletes' muscle mass and strength was significantly lower than the younger subjects.

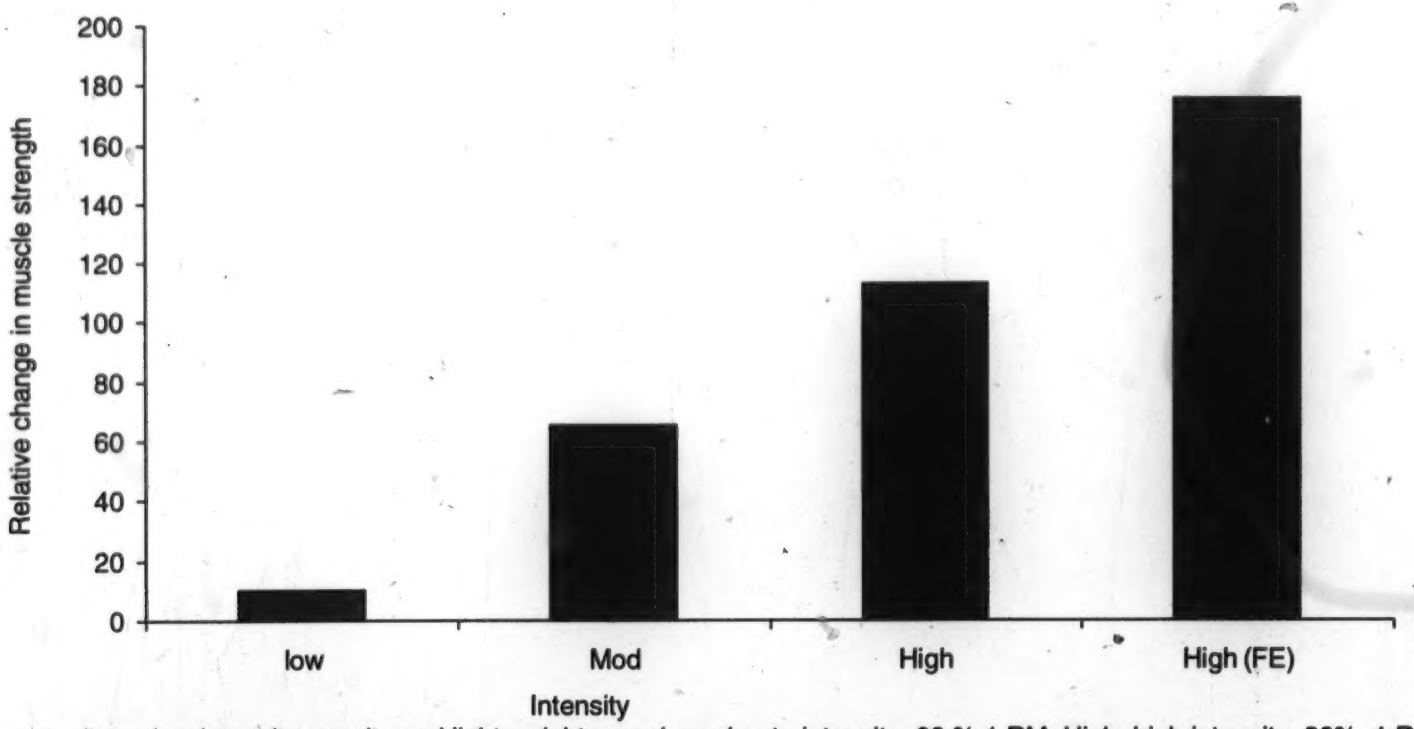
Fiatarone and co-workers (1990) have shown that muscle strength and mass can be improved into the tenth decade of life. For example, one to three

months of RT was associated with significant improvements in muscle mass (9.0%-11.4%) and maximal muscular strength (174%-227%) in older individuals between 50 and 96 years of age (Fiatarone, et al.,1988, Frontera, et al.,1988). In addition, a 5% increase in VO_{2max} was also found after training (Frontera, et al., 1990). Therefore, RT has gained popularity as a safe and effective intervention to improve muscle strength, muscle mass and in a select group of healthy older adults may also increase VO_{2max}.

Relationship between exercise intensity and changes in maximal muscular strength

It has recently been reported that the relative improvement in maximal muscular strength secondary to RT was related to the underlying intensity of the training stimulus (Fiatarone and Evans, 1993). For example, Figure 2.7 shows that the greatest increase in maximal muscular strength occurred when older adults performed a high-intensity (>80% 1 RM) RT program. Based on the above findings, it has recently been suggested that older adults should perform high-intensity RT to improve muscle strength and mass (Tseng, et al., 1995). Although high-intensity RT may be beneficial to increase skeletal mass and strength a recent series of studies have found that older individuals are capable of generating relatively high systolic pressures (i.e. 247 to 261 mmHg, McCartney, et al., 1993; Benn et al., 1996) when performing sub-maximal resistance exercise. Therefore, it may be possible that the RT mediated pressure load may be a potent stimulus to alter the size and systolic or diastolic function of the senescent LV. Accordingly, before RT can be classified as a safe

Figure 2.7 Relationship between resistance training intensity and relative change in muscle strength



(Low, low intensity, gravity and light weights, mod, moderate intensity, 60 % 1 RM, High, high intensity, 80%, 1 RM; High (FE), high intensity (80% 1RM) in the frail elderly. Adapted from Fiatarone and Evans, 1993).

exercise intervention for older adults further studies are required to ensure that RT does not harmfully alter the size or function of their heart.

Summary

Sub-maximal or maximal RT has been shown to be associated with acute hemodynamic alterations including increases in heart rate, systolic pressure, intra-thoracic pressure, intra-muscular pressure associated with a concomitant decline in peripheral vascular conductance, end-diastolic volume, end-systolic volume and stroke volume. A widely held belief in sport cardiology has been that the RT mediated "pressure load" is associated with a concomitant increase in LV wall stress which may be a potent stimulus to induce cardiac growth (Figure 2.3). However, the heightened pressure load associated with RT has been shown to be secondary to the increased intra-thoracic pressure associated with performing a brief Valslava maneuver (Lentini, et al., 1993). Previous studies have demonstrated that positive swings in intra-thoracic pressure (i.e., a brief Valsalva maneuver without lifting) were transmitted directly to the arterial vasculature as an increase in systolic pressure, however, the pressure to which the heart was "exposed" was not elevated above resting values (Hamilton, et al.,1944; Buda, et al., 1979). Therefore, it may be possible that RT performed with a brief Valsalva maneuver may reduce the pressure to which the heart is "exposed" and as a consequence may minimize the elevation in LV wall stress. Further studies are required to assess the acute effects of RT on LV wall stress.

A series of cross-sectional echocardiographic studies have demonstrated that RT may be associated with alterations in LV morphology including increases

in ventricular septal wall thickness, posterior wall thickness, relative wall thickness, and estimated LV mass (Fagard, 1996). However, a limitation of these studies has been that the athletes were younger individuals (< 25 years) who had been training for a relatively short period of their life (< 5 years). Therefore, the effects long-term RT (>10 years) and the associated pressure load on LV dimensions and mass are not well known. In addition, the upper limits of LV wall thickening secondary to long-term training require further study.

Resistance training has recently gained popularity as a safe and effective exercise intervention for older individuals to offset the age-related decline in muscle mass and strength (Fiatarone and Evans, 1993). Despite these benefits, a recent series of studies have demonstrated that healthy older adults are capable of generating relatively high systolic pressures (i.e., 241 to 261 mmHg) when performing sub-maximal RT (McCartney, et al., 1993; Benn et al., 1996). Therefore, it may be possible that the RT mediated "pressure load" may be a potent stimulus to alter the size and function of the senescent heart. Accordingly, before RT can be classified as a "safe" exercise intervention for older adults further studies are required to ensure that this type of exercise does not harmfully alter the size and systolic or diastolic function of the senescent heart.

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Chapter III

Left Ventricular Wall stress during leg-press resistance exercise

Abstract

The objective of this study was to assess the effects of leg-press exercise performed with a brief Valsalva maneuver on left ventricular (LV) systolic function and LV end-systolic wall stress in five healthy males (mean ± S.D., age: 27.6 ± 2.9 years). The subjects performed sub-maximal (80% one repetition maximum [1RM]: 337.9 ± 109.1 kg; 95% 1 RM: 400.6 ± 129.8 kg) and maximal leg-press exercise (420 ± 118.6 kg) during which central arterial pressure, intra-thoracic pressure and two-dimensional echocardiographic analysis of LV systolic function were measured. The results revealed that sub-maximal and maximal leg-press exercise was associated with a significant increase in peak systolic pressure (Rest: 146.5 ± 15.2 mmHg; 80% 1RM: 297.0 ± 10.7 mmHg; 95% 1RM: 307.3 ± 20.6 mmHg; 100%: 290.2 ± 24.0 mmHg), end-systolic pressure (Rest: 122.8 ± 12.2 mmHg; 80% 1RM: 251.2 ± 12.5 mmHg; 95% 1RM: 261.8 ± 16.4 mmHg; 100%: 247.8 ± 16.7 mmHg), diastolic pressure (Rest: 92.1 ± 9.1 mmHg; 80% 1RM: 226.0 ± 11.5 mmHg; 95% 1RM: 238.0 ± 16.3 mmHg; 100%: 220.6 ± 16.5 mmHg) and intra-thoracic pressure (rest:1.7 ± 2.9 mmHg; 80% 1RM: 111.7 ± 20.2 mmHg; 95% 1RM: 112.2 ± 21.1 mmHg; 100%: 111.0 ± 21.3 mmHg) with no change in LV end-systolic transmural pressure (Rest: 118.3 ± 12.6 mmHg; 80% 1RM: 140.0 ± 6.1mmHg; 95% 1RM: 143.1 ± 16.1mmHg; 100%: 131.8 ± 29.7 mmHg), LV end-systolic wall stress (Rest: 91.7 ± 20.2 kilodynes/cm^2; 80% 1RM: 78.0 ± 24.4 kilodynes/cm^2; 95% 1RM: 81.4 ± 25.3 kilodynes/cm^2;100%: 85.9 ± 20.1 kilodynes/cm^2) or LV fractional area change (Rest: 0.48 ± 0.03; 80% 1RM: 0.52 ± 0.11; 95% 1RM: 0.53 ± 0.06; 100%: 0.52 ± 0.05). These findings suggest that heavy leg-press exercise performed with a brief Valsalva maneuver was not associated with an alteration in LV wall stress or LV systolic function in healthy young males.

Sub-maximal or maximal resistance exercise has been associated with a transient abrupt elevation in systolic and diastolic blood pressure (i.e., 223-480/156-350 mmHg)(MacDougall, et al., 1985; Del Torre, et al., 1990). Currently, it is believed that the heightened pressure load and LV wall stress during resistance exercise may be a potent stimulus to increase LV wall thickness and mass (Figure 2.3 in review section; Colan, et al., 1987; Pelliccia, et al., 1993). However, a series of studies have demonstrated that resistance training was not associated with changes in LV dimensions and mass (Brown, et al., 1987; Elias, et al., 1991). Therefore, it may be possible that the resistance mediated pressure load and LV wall stress was of an insufficient magnitude or too brief in duration to alter LV morphology.

Since LV wall stress is directly related to LV transmural pressure (i.e. intra-vascular pressure minus intra-thoracic pressure) and LV geometry (Figure 3.1), alterations in either variable may affect LV wall stress. For example, Lentini et al., (1993) have shown that the heightened pressure load during resistance exercise was secondary to the increased intra-thoracic pressure associated with performing a brief Valsalva maneuver. However, the pressure to which the heart was "exposed" (i.e., LV transmural pressure) was lower than that predicted by the intra-vascular pressure alone. Therefore, positive swings in intra-thoracic pressure during resistance exercise may reduce the pressure to which the heart is "exposed" and as a consequence may attenuate the stimulus for LV hypertrophy (Lentini, et al., 1993).

Galanti et al., (1992) have recently demonstrated that acute alterations in LV geometry can also affect LV wall stress independent of changes in intra-vascular pressure. In their study, LV end-systolic wall stress was not altered during isometric exercise (compared to basal measures) despite a 25% increase in intra-vascular pressure. The attenuated exercise wall stress was secondary to the LV altering its shape in such a manner that the increased pressure was dissipated over a thicker LV wall and as a consequence reduced the tension per cardiac fiber.

Based on the findings of Galanti and Lentini and their co-workers (1992; 1993), it may be possible that acute heart-lung interactions and intrinsic changes in LV geometry may occur during resistance exercise to minimize LV wall stress. However, at the present time, the acute effects of heavy resistance exercise on LV wall stress have not been well studied.

The purpose of this study is to determine the effects of heavy leg-press exercise on LV end-systolic wall stress and LV systolic function in young healthy males. The hypothesis of this study is that leg-press exercise will not be associated with an alteration in LV wall stress or LV systolic function.

Methods

Subjects

The subjects for this study were five healthy males (mean ± S.D. age: 27.6 ± 2.9 years; height: 175.5 ± 7.3 cm; weight: 79.2 ± 6.4 kgs). Four subjects were experienced resistance trained athletes (3 powerlifters and 1 rower) while the remaining subject performed recreational resistance training. Ethical

approval for this study was obtained from the University of Alberta Faculty of Medicine ethics committee for human experimentation and informed consent was obtained prior to study participation.

Maximal muscular strength

Maximal muscular strength was assessed as a voluntary one-repetition maximum (1RM) using the bilateral inclined leg-press exercise. After a suitable warm-up, the subjects performed a light set of 8-10 repetitions. The subjects then completed subsequent 2 repetition sets of increasing load until only one repetition could be performed despite attempting a second repetition. This testing protocol usually required a total of 5 to 7 sets. The Leg-press exercise was standardized so that all subjects lowered the weight to a knee joint angle of 90 degrees before initiating the concentric phase of the lift. Three to five minutes of rest was allowed between sets and verbal encouragement was constantly provided. All subjects returned to the research laboratory for a second session during which time they performed sub-maximal (80% and 95% 1RM) and maximal leg-press exercise with hemodynamic, intra-thoracic and echocardiographic monitoring.

Central arterial pressure monitoring

Central arterial pressure was measured with a 5 french Millar pressure-tip transducer (Millar instruments, Houston, Texas). After infiltrating the skin with xyolocaine the left brachial artery was cannulated with a 5 French vascular sheath using the Seldinger technique. The pressure-tip transducer was then positioned in the descending aorta at the level of the left ventricle and the signal

was amplified (Electronics for Medicine, Torrance, Calif) and stored digitally on a 4 channel recorder (Quinton, EP LAB, Bothell, Wash). The pressure transducer was calibrated at baseline and before and after every leg-press set.

Intra-thoracic pressure monitoring

Intra-thoracic pressure (ITP) was measured with a 5 french Millar-pressure tip transducer (Millar Instruments, Texas) which was inserted orally and positioned in the mid-esophagus. The transducer was attached to a control unit (TCB 500, Millar instruments, Texas) and the signal was amplified (Electronics for Medicine, Torrance, Calif) and stored digitally on a 4 channel recorder (Quinton EP LAB, Bothell, Wash). The pressure transducer was calibrated at baseline and before and after every exercise set.

Left ventricular imaging

Left ventricular imaging was performed with a commercially available ultrasound instrument (Hewlett Packard, Sonos 2500) with a 5MHz transesophageal transducer. Prior to oral intubation, the gag reflex was abolished with oral Xylocaine gargle. The transesophageal probe was then inserted through the mouth and positioned into the stomach. Left ventricular 2-dimensional images were obtained from the transgastric short-axis view at the level of the mid-papillary muscles and were averaged over three cardiac cycles to obtain the following measures: (1) end-diastolic cavity area (EDCA); (2) end-systolic cavity area (ESCA); (3) end-systolic total cavity area (ESTA); (4) end-systolic myocardial area (ESMA, measured as ESTA minus ESCA) (Figure 3.1)

Calculations

Simultaneous LV cavity areas, central arterial pressure and intra-thoracic pressure measures were obtained at rest and during the last repetition of the 85% and 95% 1 RM sets as well as during the maximal leg-press set and were used to calculate the following:

(1) LV end-systolic transmural pressure (LVESTMP)

LVESTMP (mmHg) = *LVESP minus ITP

*(In the absence of aortic stenosis or LV outflow tract obstruction dicrotic central arterial pressure has been shown to approximate LV end-systolic pressure [Grossman, et al., 1977]. Since none of our subjects had aortic stenosis or LV outflow obstructions, dicrotic central arterial pressure was used as a surrogate for LV end-systolic pressure).

(2) Left ventricular systolic function was measured as fractional area change:

FAC = EDCA - ESCA/EDCA

(3) Left ventricular stroke area (LVSA) = EDCA – ESCA

(4) Left ventricular contractility = LVESTMP/ESCA

(5) LV end-systolic meridional wall stress (LVESWS)

LVESWS (Kilodynes/cm^2) = 1.33 X LVESTMP X (ESCA/ESMA)

Statistical Analysis

Comparsion of all measured variables between rest and leg-press exercise was performed with a repeated measures analysis of variance using Statistica software. If a significant difference was found then a post-hoc Scheffe test was performed. The alpha level was set "a priori" at $p < 0.05$.

Results

The brachial arterial catheterization, esophageal and transesophageal oral intubations were not associated with any complications. In addition, all subjects tolerated the exercise protocol without any side effects. Due to technical difficulties, intra-thoracic pressure was not obtained during the 95% and 100% sets in one subject. In addition, complete data was not obtained in another subject and therefore was excluded from the analysis.

Maximal bilateral leg-press 1 RM

The subjects maximal bilateral leg-press 1RM was equivalent to 420 ± 118.6 kg. During the pressure measurement exercise session, the subjects lifted 337.9 ± 109.1 kg for 9.3 ± 2.2 repetitions (80% 1RM) and 400.6 ± 129.8 kg for 4 ± 0 repetitions (95% 1 RM) as well as the above 1RM set.

Acute hemodynamic responses during leg-press exercise

Compared to rest, sub-maximal and maximal leg-press exercise was associated with significant increases in heart rate (rest: 96.0 ± 8.1 beats/min; 80% 1RM: 166.0 ± 23.6 beats/min; 95% 1RM: 150.3 ± 22.7; 100%: 135.0 ± 6.4 beats/min), peak systolic pressure (Rest: 146.5 ± 15.2 mmHg; 80% 1RM: 297.0 ± 10.7 mmHg; 95% 1RM: 307.3 ± 20.6 mmHg; 100%: 290.2 ± 24.0 mmHg), end-systolic pressure (Rest: 122.8 ± 12.2 mmHg; 80% 1RM: 251.2 ± 12.5 mmHg; 95% 1RM: 261.8 ± 16.4 mmHg; 100%: 247.8 ± 16.7 mmHg), diastolic pressure (Rest: 92.1 ± 9.1 mmHg; 80% 1RM: 226.0 ± 11.5 mmHg; 95% 1RM: 238.0 ± 16.3 mmHg; 100%: 220.6 ± 16.5 mmHg) and intra-thoracic pressure (rest: 1.7 ± 2.9 mmHg; 80% 1RM: 111.7 ± 20.2 mmHg; 95% 1RM: 112.2 ± 21.1 mmHg;

100%: 111.0 ± 21.3 mmHg). Left ventricular end-systolic transmural pressure was not significantly altered during leg-press exercise compared to rest and was always significantly lower than the simultaneous end-systolic pressure during exercise (Figure 3.2).

Effects of leg-press exercise on left ventricular cavity areas and systolic function

Left ventricular end-diastolic and end-systolic cavity areas were significantly smaller during sub-maximal and maximal leg-press exercise compared to rest (Table 3.1). However, no significant change was found for end-systolic myocardial area, end-systolic cavity to myocardial area ratio, LV contractility, fractional area change or LV end-systolic wall stress (Table 3.1 and figure 3.3)

Discussion

The major finding of this study was that leg-press exercise performed with a brief Valslava maneuver was not associated with an alteration in LV systolic function and wall stress. These findings support the study hypothesis.

Effects of leg-press exercise on heart rate, intra-thoracic pressure, end-systolic pressure and LV end-systolic transmural pressure

The finding that leg-press exercise was associated with extreme elevations in heart rate, systolic pressure, diastolic pressure and intra-thoracic pressure is similar to that previously reported for younger individuals (MacDougall, et al., 1985; 1992; Del Torre, et al., 1990; Lentini, et al., 1993). In addition, the finding that heart rate was higher during sub-maximal compared to

maximal resistance exercise was similar to that previously reported (Fleck and Dean, 1987; Falkel, et al., 1992). This finding was related to our subjects being able to perform a greater number of repetitions during sub-maximal leg-press exercise which via central "feed-forward" reflexive neural mechanisms results in an increase in sympathetic output and a concomitant elevation in heart rate (Mitchell, 1990).

The mechanism responsible for our hypertensive blood pressure response appears to be secondary to the increased intra-thoracic pressure associated with performing a brief Valsalva maneuver. For example, Figure 3.2 shows that LV end-systolic pressure was higher during leg-press exercise compared to resting measures, however LV end-systolic transmural pressure (i.e., LV end-systolic pressure minus intra-thoracic pressure) was not elevated above baseline values. This finding is consistent with earlier studies that demonstrated that positive swings in intra-thoracic pressure (i.e., a Valsalva maneuver without lifting) were transmitted directly to the arterial vasculature as an increase in systolic pressure, however, the pressure to which the heart was "exposed" (i.e., LV end-systolic transmural pressure) was not elevated above resting measures (Hamilton, et al., 1944; Buda, et al., 1979). Therefore, the heart, unlike the peripheral arteries, is not exposed to the heightened systolic pressure during a Valsalva maneuver performed alone or when incorporated during leg-press exercise (Hamilton, et al., 1944; Buda, et al., 1979; Lentini, et al., 1993).

Effects of leg-press exercise on left ventricular systolic function

The effect of heavy resistance exercise performed with a brief Valsalva maneuver on LV systolic function has not been well studied. Lentini et al., (1993), using transthoracic echocardiography, found that leg-press exercise was associated with a decline in end-diastolic volume, end-systolic volume and stroke volume. Despite the attenuated cardiac volume response, LV contractility was higher during leg-press exercise resulting in a concomitant elevation in LV systolic function. Our finding that leg-press exercise was associated with a decline in LV end-diastolic cavity area, end-systolic cavity area and stroke area with a concomitant increase in LV contractility and systolic function is nearly identical to that of Lentini and associates (1993).

The mechanism(s) responsible for our reduced LV end-diastolic cavity area may be related to the increased intra-thoracic pressure associated with performing a brief Valsalva maneuver. For example, increases in intra-thoracic pressure result in a concomitant elevation in the downstream pressure for venous return (i.e. right atrial pressure) and as a consequence may reduce venous return. However, Harman and Del Torre and their co-workers (1998; 1990) have shown that intra-abdominal pressure was always higher than intra-thoracic pressure during leg-press exercise (Figure 3.4), therefore, the positive trans-diaphragmatic pressure gradient (i.e., 31 to 42 mmHg) during this form of exercise may transiently increase venous return.

If right ventricular preload does increase during leg-press exercise then our finding of a decline in LV end-diastolic cavity area (LV preload) is of special

interest. The paradoxical finding that increases in right ventricular preload may be associated with a concomitant decrease in LV preload may be explained by diastolic ventricular interaction (Figure 3.5). For example, Bove et al., (1981) and Weber, et al., (1981) have demonstrated that increases in right ventricular volume shift the ventricular septum towards the LV resulting in a reduction in the ventricular septal to free wall dimension. The consequence of the septal shift is that LV diastolic filling may be impaired. Although we did not assess diastolic function, a series of studies have reported that weightlifters have impaired diastolic filling patterns (i.e., reduced early diastolic peak flow velocity associated with a concomitant increase in late diastolic flow velocity resulting in a reduced early to late flow velocity ratio) when performing isometric exercise (Sagiv et al., 1992; Abinader, et al., 1996). Therefore, it is conceivable that transient increases in right ventricular preload during leg-press exercise may cause a leftward shift of the ventricular septum resulting in a decline in LV preload and (in accordance with the Frank-Starling mechanism) a concomitant reduction in LV stroke area (or stroke volume).

Despite the complex heart-lung and ventricular interactions, the present finding that LV systolic function was not altered during leg-press exercise is consistent with that previously reported (Lentini et al., 1993). Together, these findings suggest that heavy leg-press exercise performed with a brief Valsalva maneuver will not likely result in an acute deterioration in LV systolic function in healthy young males.

Effects of leg-press exercise on left ventricular end-systolic wall stress

The major finding of this study was that leg-press resistance exercise was not associated with an alteration in LV end-systolic wall stress (Figure 3.3). This finding is consistent with two earlier studies that reported that athletes with physiological hypertrophy had reduced resting or exercise LV end-systolic wall stress (Colan, et al., 1987; Galanti, et al., 1992). Colan and co-workers (1987) assessed resting LV end-systolic wall stress in resistance-trained athletes and age-matched sedentary individuals. Despite the athletes' higher intra-vascular pressures, their calculated LV wall stress was lower than the non-trained subjects. The attenuated wall stress was secondary to the increased LV wall thickness which effectively reduced the pressure per cardiac fiber (Colan, 1987). Galanti and co-workers (1992) confirmed and extended these findings. In their study, they assessed the acute LV end-systolic wall stress response during sub-maximal (30% maximal voluntary contraction) isometric handgrip exercise in athletes and sedentary individuals. Compared to rest, isometric exercise was associated with a significant increase in intra-vascular pressure (~25%) in both groups. However, LV wall stress was not altered in the athletes' during isometric exercise (compared to rest) while it increased by 28% in the sedentary subjects. The attenuated end-systolic wall stress was secondary to the athletes' LV altering its geometry (i.e., decreased end-systolic cavity dimension to end-systolic wall thickness ratio) during exercise such that a larger end-systolic wall thickness was able to reduce the force (pressure) per cardiac fiber.

The mechanism(s) responsible for our finding of no change in LV end-systolic wall stress during heavy leg-press exercise are twofold. First, leg-press exercise was not associated with an alteration in LV end-systolic transmural pressure. Therefore, the pressure to which the heart was "exposed" during this form of exercise was similar to that measured at rest. Second, and not unlike the findings of Galanti et al., (1992), the results indicate that the heart alters its geometry (i.e., reduced LV end-systolic cavity to myocardial area ratio) such that the tension per cross-sectional area of the myocardium was reduced during leg-press exercise. Therefore, it appears that transient alterations in LV geometry occur during sub-maximal isometric or heavy leg-press resistance exercise to minimize LV end-systolic wall stress.

A limitation of the present study was that esophageal pressure was measured as a surrogate for intra-thoracic pressure. However, Kingma et al., (1987) have shown that esophageal pressure underestimates pericardial pressure (the "true" surrounding pressure of the heart) by 33% during positive swings in intra-thoracic pressure. Therefore, the actual LV end-systolic transmural pressure and concomitant LV end-systolic wall stress during leg-press exercise would have been lower than our reported values.

In summary, the major finding of this study was that leg-press resistance exercise performed with a brief Valsalva maneuver was not associated with acute alterations in LV end-systolic wall stress or LV systolic function.

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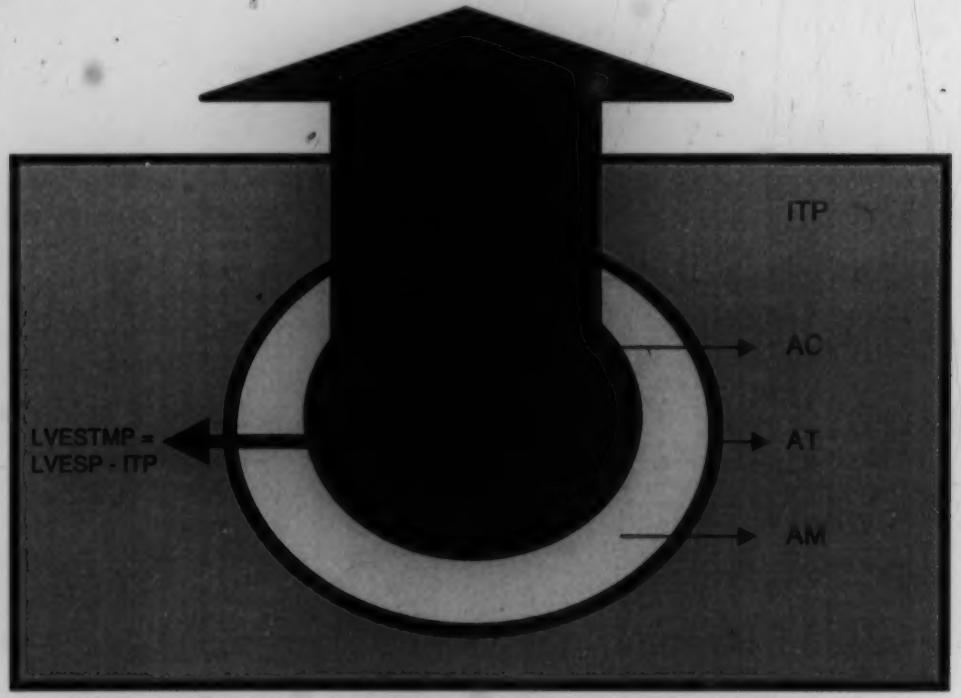
Table 3.1 Effects of leg-press resistance exercise on left ventricular cavity areas, contractility and systolic function

Variable	Rest	80%	95%	100%
EDCA (cm^2)	22.2 ± 1.4	16.7 ± 4.6*	16.5 ± 5.2*	17.3 ± 4.3
ESTA (cm^2)	32.2 ± 2.7	27.1 ± 3.6*	27.7 ± 2.9*	26.8 ± 4.4*
ESCA (cm^2)	11.6 ± 1.2	7.8 ± 1.4*	7.6 ± 2.2*	8.3 ± 2.1*
ESMA (cm^2)	20.6 ± 1.9	19.3 ± 3.1	20.1 ± 1.9	18.5 ± 2.8
ESCA/ESMA	0.57 ± 0.06	0.41 ± 0.09	0.38 ± 0.12	0.45 ± 0.08
LVSA	10.6 ± 0.8	8.9 ± 4.0	8.9 ± 3.4	9.0 ± 2.5
ESTMP/ESCA ($mmHg/cm^2$)	10.2 ± 1.7	17.7 ± 3.8	18.0 ± 5.4	15.3 ± 5.8
FAC	0.48 ± 0.03	0.52 ± 0.11	0.53 ± 0.06	0.52 ± 0.05

(Mean ± S.D.; EDCA, end-diastolic cavity area; ESTA, end-systolic total area; ESCA, end-systolic cavity area; ESMA, end-systolic myocardial area; ESCA/ESMA, end-systolic cavity to myocardial area ratio; LVSA, left ventricular stroke area; ESTMP/ESA, LV contractility; FAC, fractional area change; *, $P < 0.05$ versus rest)

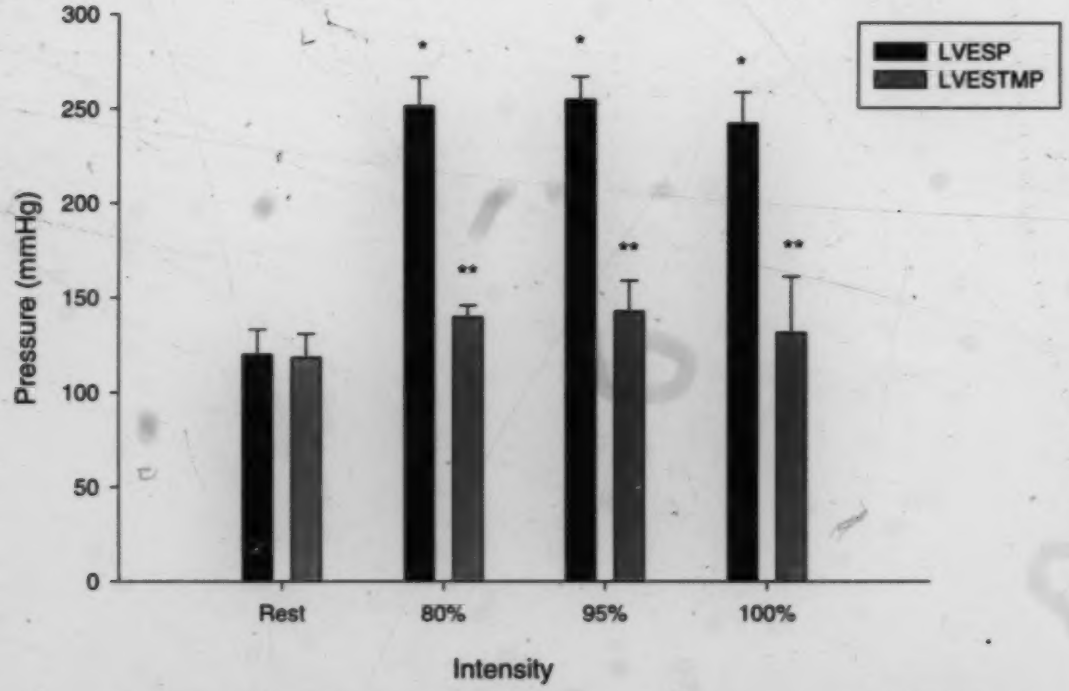
Figure 3.1 Determinants of left ventricular end-systolic wall stress



LVESWS = LVESTMP x LV geometry (AC/AM)

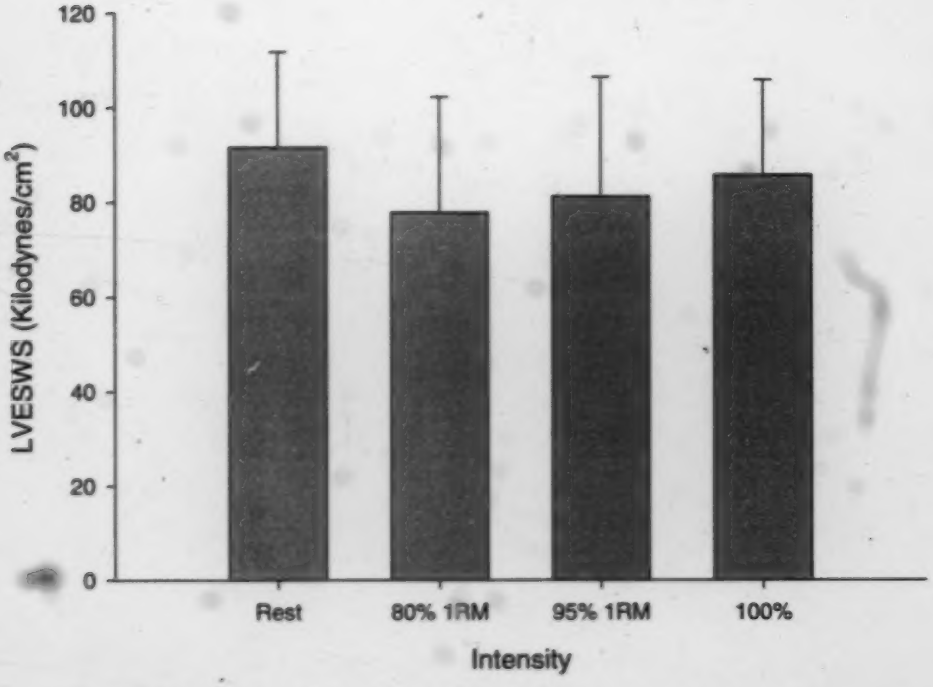
(LVESWS, left ventricular end-systolic wall stress; ITP, intra-thoracic pressure; AC, end-systolic cavity area; AM, end-systolic myocardial area; AT, end-systolic total cavity area; LVESP, left ventricular end-systolic pressure; LVESTMP, left ventricular end-systolic transmural pressure)

Figure 3.2 Effects of leg-press exercise performed with a brief Valsalva maneuver on left ventricular end-systolic pressure and left ventricular end-systolic transmural pressure



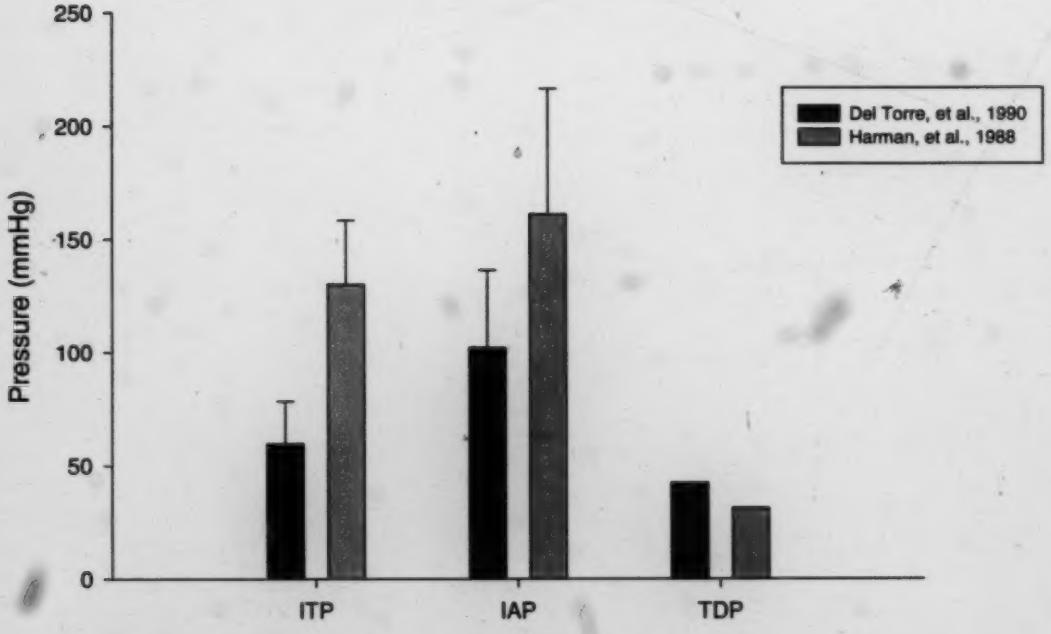
(Mean +/- S.D.; LVESP, left ventricular end-systolic pressure; LVESTMP, left ventricular end-systolic transmural pressure; *, $P < 0.05$ vs. rest; **, $P < 0.05$ vs. LVESP)

Figure 3.3 Left ventricular end-systolic wall stress during leg-press resistance exercise performed with a brief Valsalva maneuver



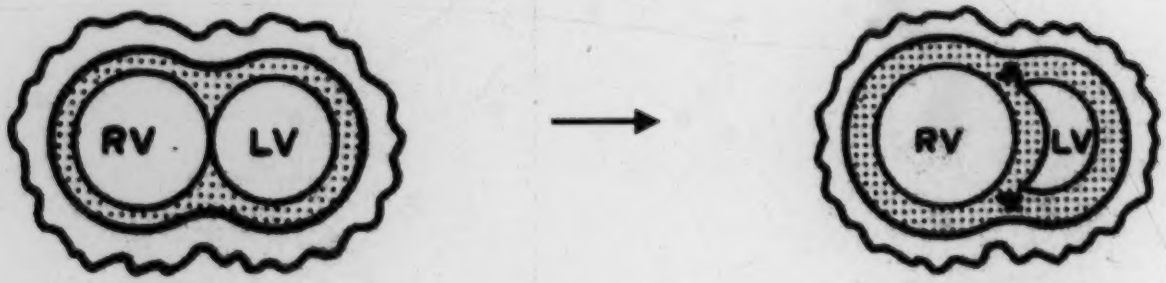
(Mean +/- S.D.; LVESWS, left ventricular end-systolic wall stress; all comparisons p >0.05)

Figure 3.4 Effects of leg-press resistance exercise on intra-thoracic pressure, intra-abdominal pressure and trans-diaphragmatic pressure



(Mean +/- S.D; ITP, intra-thoracic pressure; IAP, intra-abdominal pressure; TDP, trans-diaphragmatic pressure)

Figure 3.5 Effects of increased right ventricular end-diastolic volume on left ventricular preload: Role of diastolic ventricular interaction



Rest

Leg-press exercise

Increases in right ventricular diastolic volume can shift the ventricular septum leftwards which may impair left ventricular diastolic filling resulting in a decline in left ventricular preload

Chapter IV

Effects of long-term Resistance Training on Left Ventricular Morphology

Abstract

The objective of this study was to assess the effects of long-term (≥ 10 years) resistance training (RT) on left ventricular (LV) dimensions and mass. The study participants consisted of 21 elite male powerlifters ([RT], mean ± SD, age: 33.4 ± 5.9 years) and 10 sedentary males ([CT], age: 30.9 ± 4.2 years). Two-dimensionally guided transthoracic m-mode echocardiograms were obtained at rest to quantify LV diastolic cavity size, posterior wall thickness, ventricular septal wall thickness and LV mass. The results revealed that long-term RT was not associated with an alteration in LV diastolic cavity size (RT: 54.4 ± 4.3 mm vs. CT: 51.8 ± 5.6mm), ventricular septal wall thickness (RT: 9.73 ± 0.95mm vs. CT: 10.13 ± 0.68mm), posterior wall thickness (RT: 9.59 ± 1.46 mm vs. CT: 9.27 ± 1.43 mm) or LV mass (RT: 200.3 ± 32.5g vs. CT: 186.5 ± 40.0g). In addition, no RT athlete was found to have a LV mean wall thickness above normal clinical limits (≤ 12mm). These findings suggest that long-term RT as performed by elite male athletes does not alter LV morphology.

Key words: Resistance training; Athletes heart; Echocardiography

Resistance training (RT) has gained popularity as a safe and effective exercise intervention to increase skeletal muscle mass and muscle strength (Fiatarone and Evans, 1993). Despite the benefits of RT on skeletal muscle morphology the effects of this type of training on left ventricular (LV) morphology remains uncertain. Some studies have found that the RT mediated pressure load (i.e., systolic pressures > 250 mmHg, MacDougall, et al., 1985, 1992) may result in alterations in LV morphology including increases in LV wall thickness, relative wall thickness and estimated LV mass (Colan, et al., 1987; Deiligiannis, et al., 1988; Fagard, 1996). However, other studies have reported that RT was not associated with a change in LV dimensions or mass (Brown, et al., 1987; Elias, et al., 1991) leading to the suggestion that the heightened pressure load was too brief in duration to alter LV morphology (Rost, 1982; Shapiro, 1987).

Previous echocardiographic investigations assessing the "athletes heart" have shown that the magnitude of the alteration in LV morphology was related to the duration of athletic conditioning (Nishimura, et al., 1980; Spirito, et al., 1994; Pelliccia, et al., 1997). However, a limitation of the studies assessing the effects of RT on LV morphology was that the subjects were younger athletes (< 25 years) who had been training for a relatively short period of their life (< 5 years). Therefore, the effects of long-term (> 10 years) RT on LV morphology has not been well studied. Accordingly, the purpose of this study is to evaluate the effects of long-term RT on LV dimensions and mass in elite male RT athletes. The hypothesis of this study is that long-term RT will not be associated with an alteration in LV dimensions or mass.

Methods

Subjects

The study group consisted of 21 RT athletes who were members of the Canadian Powerlifting Union and 10 sedentary control subjects (CT). The RT athletes had been training for 10 ± 5 years and had qualified and competed at the 1996 Canadian National Powerlifting Championships where they volunteered to participate in the study. The CT subjects were of similar age as the RT athletes and were not participating in regular exercise training. Ethical approval for this study was obtained from the University of Alberta Faculty of Physical Education and Recreation Ethics Committee for Human Experimentation and informed consent was obtained prior to study participation.

Echocardiography

Left ventricular imaging was performed with a commercially available ultrasound instrument (Hewlett Packard, Sonos 2500) with a 3.5MHz transducer. Two-dimensionally guided M-mode echocardiogram examinations were performed and measurements were obtained from the parasternal short axis view just apical to the mitral valve leaflets. The echocardiographic measures were performed in accordance with the American Society of Echocardiography guidelines (Sahn, 1978) and included: ventricular septal wall thickness (VST), posterior wall thickness (PWT) and diastolic cavity dimension (LVIDd). Relative wall thickness was measured at end diastole as 2 x (PWT/LVIDd)(Reichek and Devereux, 1982). Estimated LV mass was determined by the corrected

American Society Of Echocardiography formula as: LVM (grams) = 0.83 x $[(LVIDd + PWT + VST)^3 - (LVIDd)^3] + 0.6$ (Devereaux, 1987).

Analysis

Comparison of the echocardiographic variables between the two groups was performed with a one-way analysis of variance using Statistica software. The alpha level was set "a priori" at $p < 0.05$.

Results

No significant difference was found between the RT or CT groups for age (RT: 33.4 ± 5.9 years vs. CT: 30.9 ± 4.2 years), body surface area (RT: 2.0 ± 0.1 m^2 vs. CT: 2.0 ± 0.2 m^2), heart rate (RT: 77.8 ± 13.1 beats/min vs. CT: 76.1 ± 12.6 beats/min), systolic blood pressure (RT: 140.1 ± 18.3mmHg vs. CT: 127.8 ± 10.5mmHg), diastolic blood pressure (RT: 88.3 ± 9.9mmHg vs. CT: 84.6 ± 5.0) or mean arterial pressure (RT: 105.6 ± 11.6mmHg vs. CT: 99.0 ± 4.7mmHg).

Resistance training was not associated with alterations in absolute or relative LV diastolic cavity dimension, posterior wall thickness, ventricular septal wall thickness, estimated LV mass or relative wall thickness (Table 4.1). In addition, no RT athlete was found to have a measured LV mean wall thickness above normal clinical limits (Figure 4.1).

Discussion

The major finding of this study was that long-term RT was not associated with alterations in LV diastolic cavity size, wall thickness, relative wall thickness or estimated LV mass. These findings support the study hypothesis and are consistent with previous studies that found that RT was not associated with

changes in LV diastolic cavity size (Agati, et al., 1985; Elias, et al., 1991; Lattanzi, et al., 1992; Longhurst, et al., 1980; Pelliccia, et al., 1993; Sagiv, et al., 1997; Snoeckx, et al., 1982), ventricular septal wall thickness (Brown, et al., 1987; Longhurst, et al., 1980; Pearson, et al., 1986; Snoeckx, et al., 1982), posterior wall thickness (Fleck, et al., 1989; Menapace, et al., 1982) and estimated LV mass (Brown, et al., 1987; Snoeckx, et al., 1982). In addition, our finding that no RT athlete had a LV mean wall thickness above normal clinical limits (i.e. ≤ 12mm) is similar to that previously reported for younger (mean age: 26.3 years) RT athletes (Pelliccia, et al., 1993). Together these findings suggest that long-term RT does not alter LV dimensions and mass.

Our measured echocardiographic variables are similar to that previously reported for RT athletes (Longhurst, 1980; Pearson, et al., 1986;Pelliccia, et al., 1993;Snoeckx, et al., 1982), however, they are lower than that found by others (Colan, et al., 1987; Deligiannis, et al., 1988, 1992; Lattanzi, et al. 1992; Ricci, et al., 1982; Roy, et al. 1988; Sagiv, et al., 1997; Spataro, et al., 1985; Yeater, et al., 1996). The disparity between studies may be related to (1) the underlying use of anabolic steroids, (2) differences in the type of RT athletes studied or (3) to the performance of a brief (~2 to 3 second) Valsalva maneuver during training which may reduce the stimulus for LV hypertrophy.

The majority of studies assessing the "athletes heart" have not considered the underlying use of anabolic androgenic steroids by their athletes. Yesalis et al., (1988) and Wagman et al., (1995) have found that one-half to two-thirds of

all elite RT athletes admitted to using anabolic steroids to enhance sport performance. Supraphysiologic doses of anabolic steroids have been shown to increase maximal muscular strength, muscle mass (Bhasin, et al., 1996), LV wall thickness (Dickermann, et al., 1997; Salkie, et al., 1985; Sachtleben, et al., 1993; Urhaussen, et al., 1989; Yeater, et al., 1996), relative wall thickness (Urhaussen, et al., 1989; Yeater, et al., 1996) and estimated LV mass (Mandecki, et al., 1990; Sachtleben, et al., 1993; Yeater, et al., 1996). Moreover, one investigation found that anabolic steroid use was associated with extreme LV wall thickening (i.e., mean wall thickness of 18.3 mm, range 14.7mm to 20.9mm)(McKillop, et al., 1989). Therefore, the increased wall thickness and estimated LV mass previously reported, in some RT athletes, may be secondary to the underlying use of anabolic steroids. In the present study our RT athletes were not asked about anabolic steroid use (although they were subject to random drug testing), however, if they were using anabolic steroids our results would suggest that these drugs do not alter LV morphology, a finding that is inconsistent with previous studies.

Previous echocardiographic studies assessing the effects of RT on LV morphology have most often used bodybuilders, olympic-weightlifters or powerlifters as the subjects. Although all of these athletes perform resistance exercises to increase muscle strength and/or muscle mass their acute stroke volume and cardiac output responses during this form of training may be considerably different. For example, bodybuilders have been shown to have a higher stroke volume and cardiac output response, compared to powerlifters,

when performing resistance exercise (Flakel et al., 1992). Therefore, the heightened pressure and volume load associated with bodybuilding may be a greater stimulus to induce LV hypertrophy compared to powerlifting. Pellicia and associates (1993) confirmed that the magnitude of the alteration in LV morphology may be related to the underlying type of resistance training performed as bodybuilders were found to have larger LV dimensions and mass compared to powerlifters or olympic-weightlifters. Therefore, the disparity between our findings and those of others may be related to the different types of resistance athletes studied.

A series of studies have revealed that sub-maximal or maximal resistance exercise may be associated with a transient abrupt increase in systolic blood pressure (i.e. 270 to 480mmHg; MacDougall, 1985, 1992). A widely held belief in sport cardiology has been that the increased pressure load may be a potent stimulus to alter the size of the LV (Colan, 1992). However, MacDougall and Lentini and their co-workers (1992, 1993) have shown that the transient abrupt elevation in systolic pressure during RT was secondary to the increased intra-thoracic pressure associated with performing a brief Valsalva maneuver. Since the heart and lungs are intimately related within the cardiac fossa (Butler, 1983) positive swings in intra-thoracic pressure are transmitted directly to the arterial vasculature as an increase in systolic pressure, however, the pressure that the heart "senses" was not elevated above resting values (Hamilton, et al., 1944; Buda, 1979). Therefore, an alternate explanation for our findings is that our RT athletes may have performed a brief Valsalva maneuver during training which

could have reduced the pressure to which the heart was "exposed" (chapter III) and as a consequence may have diminished the stimulus for LV hypertrophy (Lentini, et al., 1993).

A series of studies have reported that three months of RT was associated with a rapid increase (10.9%) in LV wall thickness in younger individuals (age range: 16-27 years) (Lusiani, et al, 1986; Sagiv, et al., 1986). Therefore, it is possible that our RT athletes may have had a rapid increase in LV wall thickness soon after initiation of training. However, a limitation of this hypothesis is that in order to see a similar relative increase in LV wall thickness then our athlete's baseline measurement would have had to have been ~8.5mm. Since LV wall thickness is independently related to body surface area (Sprito, et al., 1994) it is unlikely for athletes with extremely large body surface areas ($\geq 2.0m^2$), such as the ones in our study, to have this small a measure at the outset. However, further studies are required to assess the effects of short versus long-term RT on LV dimensions and mass.

Conclusion

In summary, long-term RT was not associated with changes in LV diastolic cavity size, posterior wall thickness, ventricular septal wall thickness, relative wall thickness, or estimated LV mass. In addition, no RT athlete was found to have a mean LV wall thickness above normal clinical limits. These findings suggest that long-term RT appears to be an insufficient stimulus to alter LV morphology.

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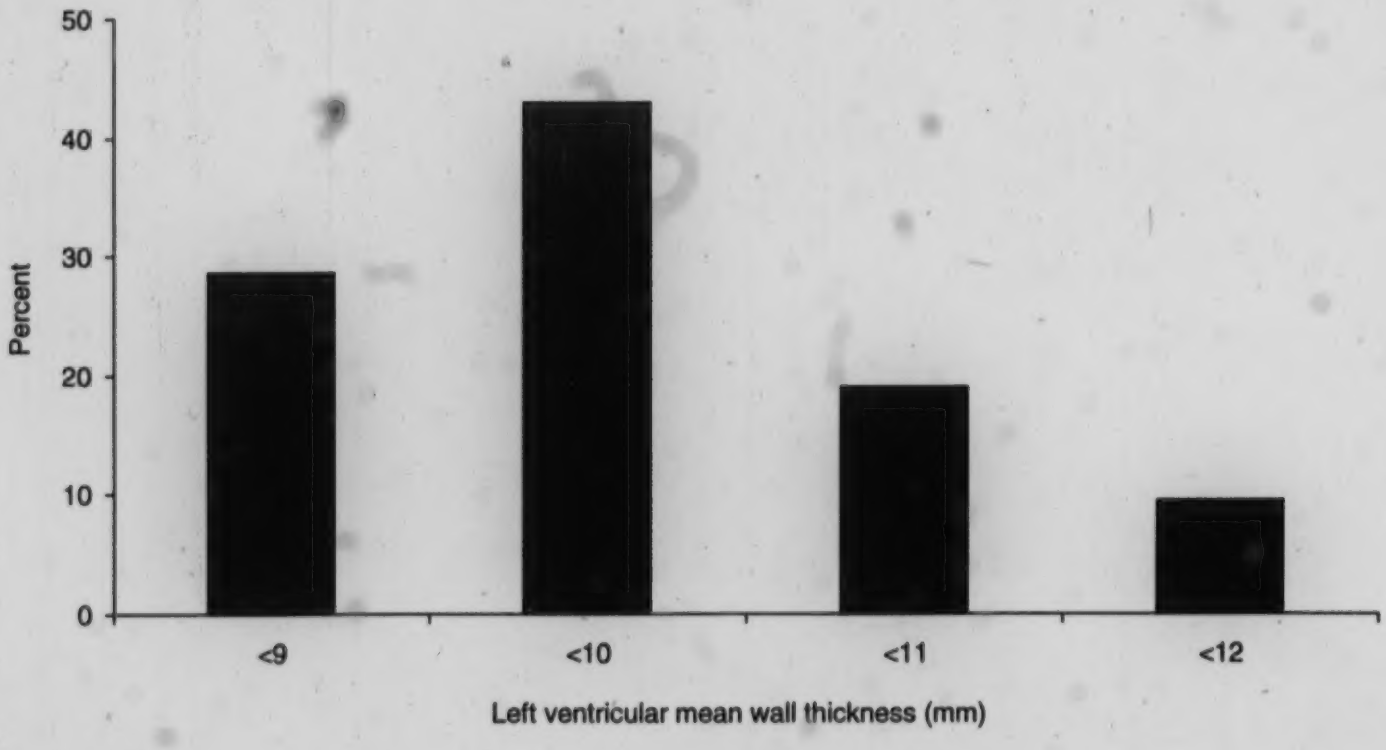
Table 4.1 Effects of long-term resistance training on Left Ventricular dimensions and mass

Variable	RT	CT
VST (mm)	9.73 ± 0.95	10.13 ± 0.68
VST (mm/m^2)	4.89 ± 0.55	5.09 ± 0.64
LVIDd (mm)	54.4 ± 4.33	51.8 ± 5.62
LVIDd (mm/m^2)	27.3 ± 2.44	25.9 ± 2.84
PWT (mm)	9.59 ± 1.46	9.27 ± 1.43
PWT (mm/m^2)	4.81 ± 0.73	4.61 ± 0.58
LVM (g)	200.3 ± 32.5	186.5 ± 40.0
LVM(g/m^2)	100.2 ± 14.5	92.7 ± 17.3
h/R	0.35 ± 0.06	0.36± 0.06

(Means ± SD; VST, ventricular septal wall thickness; LVIDd, left ventricular diastolic dimension, PWT, posterior wall thickness; LVM, left ventricular mass; h/R, relative wall thickness; all comparisons, $p > 0.05$)

Figure 4.1 Distribution of left ventricular mean wall thickness secondary to long-term resistance training



Chapter V

Left ventricular morphology in junior and master resistance trained athletes

Abstract

The objective of this retrospective cross-sectional investigation was to assess the effects of short versus long-term resistance training (RT) on left ventricular (LV) dimensions and mass. The subjects for this study consisted of 20 elite male powerlifters (8 junior athletes [JA], mean ± S.D., age: 21.1 ± 1.2 years and 12 master athletes [MA], age: 46.0 ± 5.5 years) and 19 age-matched male controls (8 young controls [YC], age: 21.8 ± 2.8 years and 11 middle-aged controls [MAC], age: 46.8 ± 4.4 years). Two dimensionally guided transthoracic m-mode echocardiograms were performed at rest to quantify LV diastolic cavity size, ventricular septal wall thickness, posterior wall thickness and LV mass. The results revealed that short or long-term RT was not associated with a significant alteration in LV diastolic cavity size (JA: 53.18 ± 4.54mm, YC: 52.13 ± 3.68mm, MA: 52.95 ± 5.06mm, MAC: 51.82 ± 4.38mm), ventricular septal wall thickness (JA: 9.45 ± 0.88mm, YC: 9.38 ± 0.92mm, MA: 9.37 ± 1.60mm, MAC: 9.73 ± 0.90mm), posterior wall thickness (JA: 9.18 ± 0.93mm, YC: 9.38 ± 0.92mm, MA, 9.03 ± 1.11mm, MAC: 9.45 ± 1.04mm) or LV mass (JA: 184.6 ± 36.1g, YC: 179.0 ± 26.5g, MA, 183.3 ± 58.1g, MAC: 184.1 ± 38.1g). These findings suggest that short or long-term RT as performed by elite junior and master athletes does not alter LV dimensions and mass.

Key words: Athletes heart, Echocardiography, Resistance training

Short or long-term athletic training has been shown to be associated with left ventricular (LV) morphologic adaptations including increases in posterior wall thickness, ventricular septal wall thickness, diastolic cavity size and estimated LV mass (Maron, 1986). The magnitude of the alteration in LV morphology has been shown to be related to the length of training exposure as master athletes had larger LV dimensions and mass compared to "sport-matched" junior athletes (Nishimura, et al., 1980; Miki, et al., 1994). However, a limitation of the studies assessing the "athletes heart" in junior and master athletes has been the primary focus on athletes performing endurance disciplines.

Previous studies have found that short-term resistance training (RT) was associated with an increase in LV wall thickness, relative wall thickness and estimated LV mass in younger individuals (Colan, et al., 1985; Deligiannis, et al., 1988). Moreover, increased age has also been shown to induce LV morphologic changes similar to that found secondary to RT (Lewis and Maron, 1992). Therefore, it may be possible that the combination of aging and RT may be a potent stimulus to induce LV hypertrophy. However, the effects of short versus long-term RT on LV morphology have not been studied.

The purpose of this retrospective cross-sectional echocardiographic study is to assess the effects of short-term (< 5 years) versus long-term RT (>10 years) as performed by junior and master athletes respectively, on LV dimensions and mass. The hypothesis of this study is that short or long-term RT as performed by junior and master athletes will not be associated with an alteration in LV dimensions or estimated LV mass.

Methods

Subjects

The subjects for this study consisted of 39 males (20 RT athletes and 19 control subjects). The RT athletes had qualified and competed at the 1995 Canadian Powerlifting Union national championships and were classified, in accordance with the International Powerlifting Federation guidelines, as junior athletes (JA, n= 8; mean age: 21.1 ± 1.2 years) or master athletes (MA, n=12; mean age: 46.0 ± 5.5 years). The control subjects were recruited from the St. Pauls' hospital cardiac echocardiography laboratory and were of similar age as the RT athletes (8 young control [YC]: mean age: 21.8 ± 2.8 years and 11 middle age controls [MAC]: mean age: 46.8 ± 4.4 years).

Assessment of LV morphology was performed at the championship meet site at a time when all athletes could be expected to be in peak physical condition (Table 5.1). Ethics approval was obtained from the University of British Columbia and St. Paul's Hospital Medical Ethics Committee and written informed consent was obtained prior to study participation.

Assessment of left ventricular dimensions and estimated mass

Left ventricular imaging was performed with a commercially available ultrasound instrument (Hewlett-Packard, Sonos 2500) with a 3.5 MHz transducer. Two-dimensionally guided m-mode echocardiogram examinations were performed using the parasternal long axis view just apical to the mitral valve leaflets. The echocardiographic measurements were performed in accordance with the American Society of Echocardiography guidelines (Sahn, et

al., 1978) and included the following: ventricular septal thickness (VST), posterior wall thickness (PWT), and diastolic internal dimension (LVIDd). Relative wall thickness (h/R) was measured as 2 x PWT/LVIDd (Reichek and Devereux, 1982). Calculated LV mass was determined by the corrected American Society of Echocardiographic formula as: LVM (g)= 0.83 x $[LVIDd+VST+PWT]^3$-$(LVIDd)^3]$+0.6. (Devereaux, 1987)

Statistical analysis

Comparison of the echocardiographic variables between the groups was performed with a one-way analysis of variance using Statistica software. The alpha level was set a priori at "$p< 0.05$".

Results

Physical characteristics

By study design, the MA and MAC were significantly older than the JA or YC (Table 5.2). However, no significant difference was found between any of the groups for body surface area (Table 5.2). The MA had significantly higher resting heart rates then the MAC or YC, however, no significant difference was found between the MA and JA for this variable (Table 5.2).

Training experience and maximal muscular strength

The MA had a significantly greater training exposure (18.3 ± 6.6 years) than the JA (4.4 ± 3.4 years). No significant difference was found between the two athletic groups for the squat, bench press or deadlift one-repetition maximums (Table 5.1).

Left ventricular dimensions and estimated mass

No significant difference was found between any of the groups for absolute or relative ventricular septal wall thickness, posterior wall thickness, diastolic cavity size, relative wall thickness or estimated LV mass (Table 5.3)

Discussion

The major finding of this study was that short or long-term RT as performed by elite junior and master athletes was not associated with an alteration in LV diastolic cavity size, posterior or ventricular septal wall thickness, relative wall thickness or estimated LV mass. These findings support the study hypothesis.

Our finding of no significant alteration in LV morphology in junior versus master athletes was similar to that previously reported for long-distance runners or triathlon athletes (Heath, et al., 1991; Douglas, et al., 1997). However, our results differ from two earlier studies that found that master endurance cyclists had larger LV dimensions and mass compared to sport-matched younger athletes (Nishimura, et al., 1980; Miki, et al., 1994). The disparity between studies may be related to the underlying duration of sport participation. For example, Nishimura and associates (1980) found that the absolute increase in LV dimensions and mass secondary to sport participation was related to the duration of the training stimulus. In their study, 60 elite cyclists were divided into three groups based on their age and training exposure (group 1: age-range: 20 to 29 years, mean training experience: 5 years; group 2: age-range: 30 to 39 years, mean training experience: 14 years; group 3, age-range: 40 to 49 years,

mean training experience: 26.5 years). Echocardiographic analysis revealed that the older athletes with three decades of training experience had significantly larger LV dimensions and estimated LV mass compared to either group of younger cyclists. However, not unlike our findings, no significant difference was found for LV wall thickness and estimated LV mass between the two younger groups with 5 to 14 years of training experience.

The findings of Nishimura et al. (1980) suggest that an extremely long duration of training (i.e., ≥ 3 decades or more) may be required for master athletes to attain larger LV dimensions and mass compared to sport-matched younger athletes. Currently, the effects of three decades of RT on LV morphology have not been well examined. However, one of our athletes (a master national and world champion) with 32 years of training experience was found to have a measured LV diastolic cavity size, posterior and ventricular septal wall thickness and estimated LV mass within normal limits (Table 5.4). Therefore, 32 years of RT may be an insufficient stimulus to alter the size of the LV.

Previous studies assessing the master "athletes heart" have found that elite middle-age or older athletes had larger LV dimensions and mass compared to age-matched sedentary individuals (Heath, et al., 1981; Gustafsson, et al., 1996). In the current study, no significant difference was found between our master athletes and age-matched controls. The disparity between our results and those of others may be related to the types of athletes studied. For example, Spiritio and associates (1994) have found that the magnitude of the

alteration in LV morphology secondary to athletic conditioning was independently related to the mode of sport participation. Endurance training had the greatest impact on increasing LV wall thickness and cavity size while RT was found to be an insufficient stimulus to result in more than a mild increase in LV wall thickness. Therefore, it may be possible that increases in LV dimensions and mass secondary to long-term exercise training may be limited to master athletes participating in endurance disciplines.

The measured echocardiographic variables, in the junior athletes, were similar to that previously reported for younger RT athletes (Brown, et al., 1987; Longhurst, et al., 1980; Lusiani, et al., 1986; Pelliccia, et al., 1993). However, the upper limit of LV wall thickening secondary to long-term training has not been well studied. Figure 5.1 demonstrates that no junior or master athlete was found to have a LV mean wall thickness above normal clinical limits (i.e. ≤ 12mm). These findings confirm and extend the results of Pelliccia and associates (1993) who found that the upper limit of LV wall thickening in younger (mean age:26.3 years) RT athletes was ≤ 12mm. Together these findings suggest that if a junior or master RT athlete is found to have a LV mean wall thickness >12 mm then further investigations may be required (to rule out other forms of pressure overload hypertrophy) as this adaptation appears to be an unlikely consequence of short or long-term RT.

Conclusions

In summary, short or long-term RT was not associated with changes in LV cavity size, wall thickness, relative wall thickness and estimated LV mass.

Moreover, the upper limit of left ventricular wall thickening with short or long-term RT does not appear to exceed normal clinical limits. Therefore, short-term or long-term RT as performed by elite junior or master RT athletes appears to be an insufficient stimulus to alter LV morphology.

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Table 5.1 Maximal squat, bench press and deadlift performed at the Canadian junior and master powerlifting championships.

Group	Sqt (Kg)	BP (kg)	DL (kg)
JA	212.8 ± 37.7	140.6 ± 19.7	229.4 ± 25.8
MA	215.8 ± 45.6	130.0 ± 32.0	218.9 ± 29.6

(Mean ± S.D.; Sqt, squat, BP, bench press; DL, deadlift; JA, junior athletes; MA, master athletes; all comparisons $P > 0.05$)

Table 5.2 Physical characteristics of the junior and master athletes and controls.

Group	Age (years)	BSA (m^2)	HR (beats/min)
JA	21.1 ± 1.2* **	1.94 ± 0.12	78.4 ± 11.2
YC	21.8 ± 2.8* **	1.91 ± 0.15	68.3 ± 8.3*
MA	46.0 ± 5.5	1.99 ± 0.26	87.2 ± 11.7**
MAC	46.8 ± 4.4	1.97 ± 0.23	71.3 ± 8.2

(Mean ± S.D.; BSA, body surface area; HR, heart rate, JA, junior athletes; YC, young controls; MA, master athletes; MAC, middle age controls; *, $p < 0.05$ vs MA; **, $p < 0.05$ vs. MAC)

Table 5.3 Left ventricular dimensions and mass in junior and master athletes and age-matched controls.

Variable	JA	YC	MA	MAC
VST (mm)	9.45 ± 0.88	9.38 ± 0.92	9.37 ± 1.60	9.73 ± 0.90
VST (mm/m^2)	4.88 ± 0.48	4.94 ± 0.67	4.70 ± 0.43	4.98 ± 0.66
LVIDd (mm)	53.18 ± 4.54	52.13 ± 3.68	52.95 ± 5.06	51.82 ± 4.38
LVIDd (mm/m^2)	27.44 ± 2.10	27.40 ± 2.50	26.94 ± 3.61	26.44 ± 2.51
PWT (mm)	9.18 ± 0.93	9.38 ± 0.92	9.03 ± 1.12	9.45 ± 1.04
PWT (mm/m^2)	4.74 ± 0.52	4.94 ± 0.67	4.56 ± 0.38	4.83 ± 0.64
LVM (g)	184.6 ± 36.1	179.0 ± 26.5	183.3 ± 58.1	184.1 ± 38.1
LVM (g/m^2)	95.0 ± 16.8	94.1 ± 15.3	91.3 ± 20.8	93.3 ± 16.2
h/R	0.35 ± 0.05	0.36 ± 0.05	0.34 ± 0.03	0.37 ± 0.04

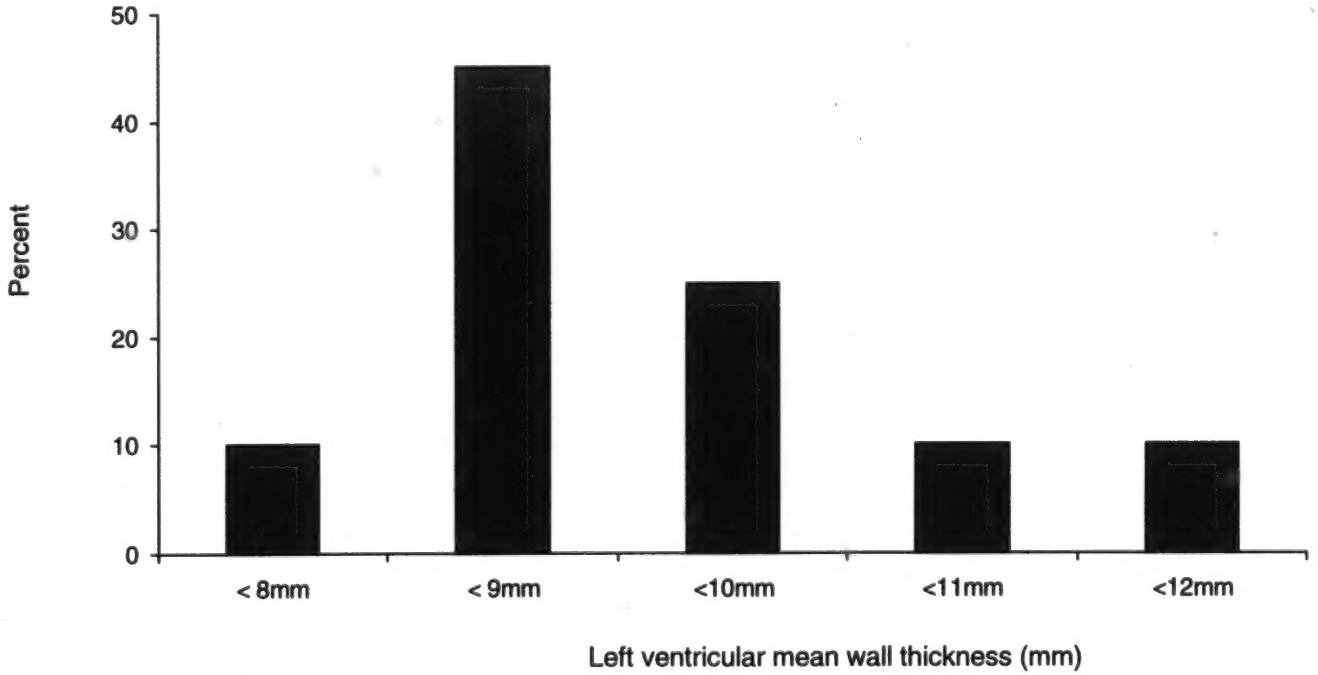
(Mean ± S.D; JA, junior athletes; YC, young controls; MA, master athletes: MAC, middle age controls; VST, ventricular septal thickness; LVIDd, left ventricular diastolic dimension, PWT, posterior wall thickness; LVM, left ventricular mass; h/R, relative wall thickness; all comparisons, $p > 0.05$)

Table 5.4 Left ventricular dimensions and estimated mass in a world class master RT athlete with 32 years of training experience

Variable	RT athlete
Age	56.0
BSA	1.78
VST	8.81
LVIDd	49.5
PWT	8.81
LVM (g)	151
LVM (g/m^2)	84.8

(VST, ventricular septal thickness; LVIDd, left ventricular diastolic cavity dimension, PWT, posterior wall thickness; LVM, left ventricular mass; Accepted normal limits of LV dimensions and mass for individuals without heart disease are as follows: VST or PWT, 8 to 12 mm; LVIDd, 40 to 52 mm; LVM , 134g/m^2 based on data from Pelliccia, et al.,1991).

Figure 5.1 Distribution of left ventricular mean wall thickness in junior and master resistance trained athletes



Chapter VI

Effects of 16 weeks of resistance training on left ventricular morphology and systolic or diastolic function in healthy older males

Abstract

The purpose of this study was to assess the effects of 16 weeks of resistance training (RT) on left ventricular (LV) morphology and systolic or diastolic function in healthy older males. After baseline assessments, the subjects were randomly assigned into a RT group (n = 10; mean ± S.D., age: 68.3 ± 3.2 years) or a non-exercise control group ([CT], n =10; age: 67.7 ± 3.7 years). Resistance training was performed 3 times per week for 16 weeks at a mean intensity between 60 to 80% (range: 50 to 90%) of one repetition maximum (1RM). Leg-press and bench-press 1RM's and two-dimensional and pulsed-Doppler echocardiograms were performed at baseline and after 4, 8, 12 and 16 weeks of training in the RT group. Compared to baseline, 16 weeks of RT was associated with a significant increase in leg-press 1RM (baseline: 284.8 ± 48.3 kg vs. Post 16 weeks: 366.9 ± 47.2 kg) and bench-press 1RM (baseline: 59.3 ± 11.1 kg vs. Post 16 weeks: 68.9 ± 10.6 kg). No significant change in leg-press 1RM (baseline: 290.9 ± 58.8 kg vs. Post 16 weeks: 289.7 ± 52.5 kg) or bench-press 1RM (baseline: 60.1 ± 9.0 kg vs. Post 16 weeks: 61.0 ± 12.5 kg) was found for the CT subjects during the same time period. In addition, RT was not associated with changes in diastolic or systolic cavity size, posterior wall thickness, ventricular septal wall thickness, relative wall thickness, estimated LV mass, LV wall stress, systolic or diastolic function after 4, 8, 12 and 16 weeks of exercise. These findings suggest that 16 weeks of RT was sufficient to increase leg-press and bench-press maximal strength but did not alter the size and systolic or diastolic function of the LV.

Resistance training (RT) has recently gained acceptance as a safe and effective exercise intervention for older adults to offset the age-related decline in muscle mass and strength (Fiatarone and Evans, 1993). Previous studies have shown that 8 to 52 weeks of RT was associated with increases in muscle mass (8.7 to 17.4%) and maximal muscular strength (32 to 174%) in older individuals between 50 and 78 years of age (Brown, et al., 1990; Frontera, et al., 1988,1990; Fiatarone, et al., 1990; McCartney, et al., 1996; Pyka, et al., 1994). Despite the benefits of RT on skeletal muscle morphology this form of training has been associated with an acute and abrupt increase in systolic pressure which may be a potent stimulus to alter the size of the left ventricle (LV)(Colan, 1992).

MacDougall and associates (1992) have found that the magnitude of the pressure load during resistance exercise was independent of the absolute weight lifted or size of the exercising muscle mass. Therefore, individuals with reduced muscle mass and strength (i.e., elderly or frail elderly) may be capable of generating a relatively high systolic blood pressure during RT (i.e., 261 mmHg)(Benn et al., 1996). At the present time the effect of the RT mediated pressure load on LV morphology and function in older adults is not well known. Therefore, before RT can be classified as "safe" for older individuals further studies are required to ensure that this form of training does not alter the size or function of the senescent LV.

The purpose of this study is to assess the effects of 16 weeks of RT on LV dimensions, estimated LV mass and systolic and diastolic function in healthy

older males. The hypothesis of this study is that 16 weeks of RT will not be associated with an alteration in LV morphology and systolic or diastolic function.

Methods

Subjects

The subjects for this study consisted of twenty-two males between 61 and 76 years of age (mean: 68.0 ± 3.4 years) who met the following inclusion criteria. 1) no clinical evidence of cardiovascular disease or hypertension (resting systolic blood pressure ≥ 140 mmHg or diastolic blood pressure ≥ 90 mmHg). 2) normal resting electrocardiogram (ECG). 3) normal ECG response to peak treadmill exercise, performed using the Bruce protocol. 4) absence of use of cardiovascular medications. 5) sedentary lifestyle as defined by the Baltimore longitudinal study on aging as no regular participation in endurance training or RT > 30 minutes of such activity > 3 times/week (Fleg, et al., 1995). 6) absence of cerebrovascular or orthopedic disability that would limit RT. 7) normal resting LV systolic and diastolic function as measured by 2-dimensional or pulsed-Doppler echocardiographic measures, respectively.

Baseline Assessments

Weight-room orientation

All subjects attended an orientation session during which time they toured the weight room facility and were given instructions regarding the proper use of the machines or free weight exercises that would be used in the study. The subjects then performed all of the resistance exercises and verbal feedback was provided regarding their technique.

Upper and lower extremity maximal muscular strength

Maximal muscular strength was assessed as a voluntary one-repetition maximum (1RM) for bilateral inclined leg-press and bench-press exercises. The subjects performed a five-minute warm up on a cycle ergometer followed by an easy set of 8 to 10 repetitions. A second set of 6-8 repetitions was performed with a heavier weight (~50 to 65% 1 RM). The subjects then completed subsequent 2 repetition sets of increasing load until only one repetition could be performed despite attempting a second repetition. This testing protocol usually required a total of 5 to 8 sets. The leg-press exercise was standardized so that all subjects lowered the weight to a knee joint angle of 90 degrees before initiating the concentric phase of the lift. The bench-press exercise was performed with the subjects lowering the bar to their chest where it remained motionless before pushing the bar to the initial starting position. Three to five minutes of rest was allowed between sets and verbal encouragement was consistently provided. After a five to seven day rest period the subjects performed a second strength test and the greatest weight lifted was used as the baseline 1RM.

Echocardiographic and pulsed-Doppler measurements

Left ventricular imaging was performed with a commercially available ultrasound instrument (Hewlett Packard, Sonos 2500) with a 3.5MHz transducer. Two-dimensionally guided M-mode echocardiogram examination was performed on each subject and measurements were obtained from the parasternal short axis views just apical to the mitral valve leaflets and were averaged over three

cardiac cycles. The echocardiographic measures were performed in accordance with the American Society of Echocardiography guidelines (Sahn, 1978) and included: ventricular septal wall thickness (VST), posterior wall thickness (PWT), diastolic and systolic internal dimensions (LVIDd, LVIDs, respectively). Relative wall thickness (h/R) was measured at end diastole as 2 x (PWT/LVIDd; Reichek and Devereux, 1982). Left ventricular systolic function was calculated as (LVIDd-LVIDs/LVIDd). Estimated LV mass was determined by the corrected American Society Of Echocardiography formula as: LVM (grams) = 0.83 x [(LVIDd + PWT +VST)^3 – (LVIDd)^3] + 0.6 (Devereaux, 1987). LV end-systolic meridional wall stress was calculated by as: WS (kilodynes/cm^2) = 0.33 x SBP x LVIDs/[(PWTs)[1 + (PWTs/LVIDs)] (Colan, et al., 1987). Diastolic function was assessed as peak early transmitral diastolic flow velocity and peak late (atrial) flow velocity which were measured in the apical four chamber view with the sample volume on the ventricular side of the mitral annulus.

Randomization

After the baseline tests the subjects were matched according to their leg-press and bench-press 1 RM scores and were randomly assigned to 4 months of RT or to a non-exercise control group (CT).

Resistance training program

Resistance training was performed three times per week for 16 weeks using a combination of free weights and machines. The following exercises were used: inclined leg press, leg extensions, calf raises, bench press, shoulder press, latissimus dorsi pulldowns, triceps pushdowns, and biceps curls. Except for the

bicep curls all exercises were performed bilaterally. The mean exercise intensity progressively increased from 60% to 80% (range: 50%-90%) 1RM throughout the four month program with the number of repetitions ranging from 3 to 10. The training programs were generated using a computer software package (B.E. Software, Lincoln, Nebraska) based on the actual 1 RM (bench-press and leg-press) and on a predicted 1 RM calculated from a multiple repetition test for the other exercises. All resistance exercises were re-tested every four weeks of the study to account for any rapid neurologic changes associated with training and the program was adjusted accordingly. Subjects randomly assigned to the control group were requested to continue their normal activities of daily living for the duration of the study. Ethical approval for this study was obtained from the University of Alberta Faculty of Medicine Ethics Committee for human experimentation and informed consent was obtained prior to baseline assessments.

Statistical analysis

Statistical analysis was performed with a two-way (group x time) analysis of variance. In addition, the time-course of LV morphology and muscle strength, in the RT group, was assessed using a one-way repeated measure analysis of variance. If a significant interaction was found then a post-hoc Scheffe test was performed. The alpha level was set "a priori" at $p < 0.05$.

Results

Twelve subjects were randomly assigned to the RT group and ten subjects were assigned to the CT group. Two subjects who were randomly

assigned to the RT group did not complete the study (one could not make the time commitment and quit after 4 weeks of training and the other sustained an injury not related to the study and dropped out after the mid-testing phase). In addition, one RT subject did not perform the final leg-press test. Two CT subjects had elective surgery during the study period (one had cataract surgery and was requested by his physician not to perform any heavy lifting therefore did not complete the final strength tests; the other subject had rotator cuff repair and was unable to complete the final bench-press test). In addition, two CT subjects did not perform the final bench-press test. The remaining 10 RT subjects completed the study without injury or adverse effects and had an overall attendance rate to the training sessions of 97%.

Baseline assessments

At the baseline, no significant difference was found between the groups for age (RT: 68.3 ± 3.2 years vs. CT: 67.7 ± 3.7 years), body surface area (RT: 2.00 ± 0.1 m^2 vs. CT: 1.95 ± 0.2 m^2), resting heart rate (RT: 60.2 ± 9.2 beats/min vs. CT: 62.9 ± 6.4 beats/min), resting systolic blood pressure (RT: 114.2 ± 10.9 mmHg vs. CT: 119.1 ± 11.5 mmHg), resting diastolic blood pressure (RT: 73.0 ± 6.3 mmHg vs. CT: 76.7 ± 7.4 mmHg), resting mean arterial pressure (RT: 86.7 ± 7.0 mmHg vs. CT: 90.8 ± 8.3 mmHg), diastolic cavity dimension (RT: 52.5 ± 3.7 mm vs. CT: 54.8 ± 3.5 mm), ventricular septal wall thickness (RT: 10.8 ± 0.9mm vs. CT: 11.0 ± 2.1 mm), posterior wall thickness (RT: 9.97 ± 1.6 mm vs. CT: 10.8 ± 1.4 mm), estimated LV mass (RT: 207.5 ± 26.7 g vs. CT: 238 .1 ± 45.5 g), LV end-systolic wall stress (RT: 52.9 ± 16.9 kilodynes/cm^2 vs. CT: 60.5 ± 22.4

kilodynes/cm^2), fractional shortening (RT: 0.38 ± .08 vs. CT: 0.38 ± .07), peak early (E) diastolic flow velocity (RT: 66.1 ± 11.2 cm/sec vs. CT: 74.3 ± 13.4 cm/sec), peak late (A) diastolic flow velocity (RT: 71.9 ± 18.9 cm/sec vs. CT: 77.4 ± 13.6 cm/sec) or peak E/A ratio (RT: 0.97 ± 0.29 vs. CT: 0.99 ± 0.25). In addition, no significant difference was found between the RT or CT subjects for maximal leg-press 1 RM (RT: 284.8 ± 48.3 kg vs. CT: 290.9 ± 58.8 kg) or bench-press 1 RM (RT: 59.3 ± 11.0 kg vs. CT: 60.1 ± 9.0 kg) prior to training.

Effects of 16 weeks of resistance training on Left ventricular morphology and peak diastolic flow velocity

In the RT subjects, no significant difference was found between the baseline, 4, 8, 12 or 16 week assessments for resting heart rate (Baseline: 60.2 ± 9.2 beats/min vs. Post 4 weeks: 55.0 ± 13.0 beats/min vs. Post 8 weeks: 58.5 ± 14.5 beats/min vs. Post 12 weeks: 61.4 ± 16.2 beats/min vs. Post 16 weeks: 58.1 ± 14.6 beats/min), resting systolic blood pressure (Baseline: 114.2 ± 10.9 mmHg vs. Post 4 weeks: 116.4 ± 9.4 mmHg vs. Post 8 weeks: 111.0 ± 11.7 mmHg vs. Post 12 weeks: 121.3 ± 9.8 mmHg vs. Post 16 weeks: 119.8 ± 11.1mmHg), resting diastolic blood pressure (Baseline: 73.0 ± 6.3 mmHg vs. Post 4 weeks: 75.2 ± 6.3 mmHg vs. Post 8 weeks: 73.2 ± 7.3 mmHg vs. Post 12 weeks: 78.4 ± 6.4 vs. Post 16 weeks: 79.0 ± 6.0), resting mean arterial pressure (Baseline: 86.7 ± 7.0 mmHg vs. Post 4 weeks: 88.9 ± 6.9 mmHg vs. Post 8 weeks: 85.8 ± 8.4 mmHg vs. Post 12 weeks: 92.7 ± 7.1 mmHg vs. Post 16 weeks: 92.6 ± 7.1 mmHg). In addition, no significant difference was found at any

testing time for LV systolic or diastolic cavity dimensions, wall thickness, estimated LV mass, LV wall stress, systolic or diastolic function (Table 6.1).

Effects of 16 weeks of resistance training on upper and lower extremity maximal muscular strength

A significant group by time interaction was found for upper and lower extremity maximal strength with sixteen weeks of RT resulting in a 16% and 29% increase in bench-press and leg-press 1RMs, respectively (Table 6.2). No significant increase in upper or lower extremity maximal muscular strength was found in the CT subjects after the same time period (Table 6.2). In addition, the leg-press and bench-press 1RM was significantly higher in the RT group compared to the CT group after 16 weeks of training (Table 6.2). Figures 6.1 and 6.2 show the time course of the alteration in leg-press and bench-press 1RM's in the RT group. Bench-press maximal muscular strength was significantly greater after 12 and 16 weeks of training compared to baseline. Leg-press maximal strength was significantly greater after 8, 12 and 16 weeks of training compared to baseline. In addition, the leg-press 1RM was significantly higher after 12 and 16 weeks of training compared to the four-week test. However, no significant differences were found between the 12 and 16-week measurements.

Main time effect (independent of group allocation) for resting hemodynamics and LV morphology

A main time effect, independent of group allocation, was observed for resting systolic blood pressure (Baseline: 116.7 ± 11.1 mm/Hg vs. Post 16

weeks: 125.3 ± 15.0 mmHg), resting diastolic blood pressure (Baseline: 74.8 ± 6.9 mm/Hg vs. Post 16 weeks: 79.7 ± 6.4 mm/Hg), resting mean arterial blood pressure (Baseline: 88.8 ± 7.7 mm/Hg vs. Post 16 weeks: 94.9 ± 8.5 mm/Hg), diastolic cavity size (Baseline: 53.7 ± 3.7 mm vs. Post 16 weeks: 51.4 ± 5.4 mm), ventricular septal thickness (Baseline: 10.9 ± 1.5 mm vs. Post 16 weeks: 10.2 ± 1.2mm), ventricular septal thickness indexed to body surface area (Baseline: 5.5 ± 0.8 mm/m^2 vs. Post 16 weeks: 5.2 ± 0.6 mm/m^2), estimated LV mass (Baseline: 222.8 ± 38.4 g vs. Post 16 weeks: 192.1 ± 39.2 g) or LV mass indexed to body surface area (Baseline: 112.7 ± 19.2 g/m^2 vs. Post 16 weeks: 96.9 ± 16.3 g/m^2) with the post 16 weeks echocardiographic measures being significantly lower than the baseline tests. However, no main time effect was found for any other measured echocardiographic or pulsed-Doppler variable (Table 6.3).

Discussion

The major finding of this study is that 16 weeks of RT is associated with significant improvements in leg-press and bench press maximal muscular strength, however, this form of training was not associated with a change in the size and systolic or diastolic function of the LV. These findings support the study hypothesis.

Effects of resistance training on maximal muscular strength

The finding that RT was associated with increases in upper and lower extremity muscular strength is similar to that previously reported (Frontera, et al., 1988; Brown, et al., 1990; Fiatarone, et al., 1990; Pyka, et al., 1994; McCartney,

et al., 1995; 1996). Furthermore, the relative increase in leg-press (29%) and bench-press (16%) 1RM's after 16 weeks of training was also similar to that previously reported for healthy older adults after a similar training duration (Brown, et al., 1990; Pyka, et al., 1994).

Time course for the alteration in muscle strength secondary to resistance training

Figures 6.1 and 6.2 demonstrate that 92% to 98% of the increase in leg-press and bench-press maximal strength occurred within 8 to 12 weeks of training. However, the leg-press and bench-press maximal strength appeared to plateau after three months of training as no significant difference was found for either strength measure between the 12 and 16 week tests. Our finding that maximal muscular strength may plateau within three months of initiation of RT, in novice lifters, is similar to that previously reported (Hakinen, et al., 1994). For example, Hakkinen et al. (1994) and Pyka et al., (1994) assessed the effects of 12 to 52 weeks of RT in older adults (61 to 78 years of age). In both studies it was found that rapid increases in muscle strength occurred within 2 months of training followed thereafter by a modest increase or a plateau in maximal strength. However, McCartney, et al. (1996) have found that leg-press and bench-press 1RM's did not plateau during a 2 year RT program in older adults. Therefore, it is also possible that our finding of no change in leg-press and bench-press strength between 3 and 4 months of training may not have been a true plateau, but rather a "transient" phase and that maximal strength may have

continued to increase had our subjects trained for a longer period of time (McCartney, et al., 1996).

A limitation of this study was that we did not assess the underlying mechanisms responsible for the increase in muscle strength. However, previous studies have attributed the increase in strength to neural mechanisms (Moritani and deVries, 1980) or to skeletal muscle hypertrophy (Frontera, et al., 1988; Fiatarone, et al., 1990).

Effects of resistance training on left ventricular morphology and systolic and diastolic function

Despite the RT program resulting in an increase in upper and lower extremity maximal muscular strength this form of training was not associated with changes in LV dimensions, mass or systolic and diastolic function. These findings are similar to that previously reported for younger or older individuals (Sagiv, et al., 1989; Brown, et al., 1987; Elias, et al., 1991). However, they differ from a series of studies that found an increase in LV wall thickness, relative wall thickness and estimated LV mass after 4 to 20 weeks of training in younger (range: 16 to 27 years) individuals (Kanakis and Hickson, 1980: Sagiv, et al., 1982; Lusiani, et al., 1986; Ricci, et al., 1982; Mandecki, et al., 1990). The disparity between studies may be due to an age-mediated diminution in the plasticity of the LV to alter its size secondary to exercise training. For example, a series of echocardiographic studies have found that 3 to 8 months of endurance or resistance exercise was not associated with changes in LV wall thickness, diastolic cavity size or estimated LV mass in previously sedentary middle-aged or

older adults (age range: 37 to 68 years)(Wolfe, et al., 1979; Ditchey, et al., 1981; Peronnet, et al., 1980; Perrault, et al., 1982; Morrisson, et al., 1986; Sagiv, et al., 1989). Contrary to the above hypothesis, Nishimura et al., (1980) and others (Miki et al., 1994) have found that the ability of the human heart to alter its size was not diminished with age as master athletes were found to have larger LV dimensions and mass compared to sport-matched younger athletes. However, in order to attain the increased dimensions and mass the master athletes had to train for a substantially longer period of time (Nishimura, et al., 1980). Therefore, it may be possible that previously sedentary older individuals who initiate a RT program later in life may require a longer training duration to induce LV hypertrophy similar to that observed in younger individuals.

Currently it is believed that the heightened hemodynamic load and LV wall stress associated with RT may be an important stimulus to increase LV wall thickness and mass (Figure, 2.3 in review chapter; Colan, 1992). Since older adults are not immune to the heightened pressure load during RT, such as the report of systolic pressures as high as 261mmHg (Benn, et al., 1996), it could be anticipated that the increased wall stress may be a potent stimulus to induce LV hypertrophy. However, the present study did not find that RT was associated with alterations in LV wall stress after 4, 8, 12 or 16 weeks of training (Table 6.1). Moreover, no RT subject was found to have a LV mean wall thickness above clinically acceptable limits after training (Figure 6.3). Therefore, the finding that LV wall stress was not altered during the 16 week regimen in accordance to the load-induced hypertrophy hypothesis suggests that the RT mediated

hemodynamic load was of an insufficient level (chapter III) or too brief in duration to alter the size of the LV (Rost, 1982). More importantly, the finding that RT was not associated with an alteration in fractional shortening suggests that this form of training is not likely to negatively alter LV systolic function in healthy older adults.

Effects of resistance training on left ventricular diastolic function

Previous studies have shown that senescence was associated with a decline in peak early diastolic flow velocity associated with a compensatory increase in peak late diastolic flow velocity resulting in a reduced early-to-late flow velocity ratio (Spirito and Maron, 1988; Lewis and Maron, 1992; Klein, et al., 1994; Fleg, et al., 1995). The underlying mechanisms responsible for the age-mediated alteration in diastolic filling patterns are not completely known. However, they do not appear to be related to underlying fitness levels as master athletes had diastolic flow velocity profiles similar to that of age-matched sedentary individuals (Fleg, et al., 1995; Douglas, and O'Toole, 1992).

Currently, there is a paucity of information characterizing the effects of RT on diastolic function. However, Colan and Pearson and their co-workers (1985,1992) have found that RT was not associated with alterations in LV diastolic function. In the present study, the peak early and late flow velocities and the early-to-late flow velocity ratio were similar to that reported for older adults (OH, et al., 1997). However, RT did not exacerbate the age-mediated decline in diastolic function as no significant difference was found for peak early or late flow velocities or their ratio after 4, 8, 12 or 16 weeks of training (Table

6.1) in the present study. These findings are in agreement with previous studies and suggest that RT as performed by younger or older individuals is not likely to deteriorate LV diastolic function.

Main time effect for left ventricular mass

Of particular interest was the finding that LV diastolic cavity size, ventricular wall thickness and estimated mass (independent of group allocation) were lower at the end of the study compared to baseline measures. Previous studies have found that increased age was associated with an increase in LV wall thickness and estimated LV mass in individuals non-selected for underlying coronary disease (Dannenberg, et al., 1989). However, when individuals were vigorously screened for underlying coronary artery disease LV cavity size, wall thickness and mass did not increase with senescence (Shub, et al., 1994). Moreover, Dannenberg and associates (1989) have shown that LV mass decreased by 6% between the third and eighth decades of life in individuals without underlying coronary artery disease, obesity or hypertension. In the current study, the subjects were vigorously screened for underlying coronary artery disease (physical examination, resting and stress ECG) and hypertension. In addition, all subjects had normal resting LV systolic function and were free from underlying wall motion or diastolic filling abnormalities. Therefore, the finding of a decline in LV dimensions and mass may be a usual age-related adaptation that occurs in "healthy" individuals without overt coronary disease. However, the absolute change in LV diastolic cavity size and wall thickness between baseline and post-test assessments are within the methodological error

of m-mode echocardiography (Wolfe, et al., 1986). Therefore, a more likely explanation for our findings was that they could be related to the inherent error of the technique used to assess LV morphology.

In summary, 4 months of RT was sufficient to increase upper or lower extremity maximal muscular strength. However, this form of training was not sufficient to alter LV systolic or diastolic cavity dimensions, mass and systolic or diastolic function. Therefore, RT may be classified as a safe and effective exercise intervention that can be performed by previously sedentary healthy older adults to improve overall muscle strength but does not seem to alter the size and systolic and diastolic function of the LV.

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Table 6.1 Effects of 16 weeks of resistance training on left ventricular dimensions, mass, systolic and diastolic function

Variable	Baseline	4 weeks	8 weeks	12 weeks	16 weeks
VSTd (mm)	10.7 ± 1.0	10.9 ± 0.9	10.1 ± 1.1	9.9 ± 0.8	9.9 ± 1.0
VSTd (mm/m^2)	5.4 ± 0.4	5.6 ± 0.5	5.1 ± 0.7	4.9 ± 0.5	5.0 ± 0.5
VSTs (mm)	15.5 ± 2.4	15.6 ± 1.8	14.8 ± 1.4	15.2 ± 1.6	15.7 ± 1.6
VSTs (mm/m^2)	7.9 ± 1.2	7.9 ± 0.7	7.6 ± 1.0	7.6 ± 0.9	8.0 ± 0.8
PWTd (mm)	10.0 ± 1.6	10.3 ± 1.0	10.3 ± 1.3	9.8 ± 1.0	9.9 ± 1.1
PWTd (mm/m^2)	5.1 ± 0.7	5.2 ± 0.4	5.2 ± 0.7	4.9 ± 0.5	5.0 ± 0.5
PWTs (mm)	16.0 ± 1.7	16.0 ± 2.2	16.3 ± 2.2	15.0 ± 2.5	15.9 ± 1.9
PWTs (mm/m^2)	7.8 ± 1.0	7.9 ± 1.1	8.2 ± 1.2	7.5 ± 1.3	7.8 ± 0.5
LVIDd (mm)	53.6 ± 3.4	52.2 ± 4.2	55.9 ± 3.8	54.3 ± 3.6	53.6 ± 4.9
LVIDd (mm/m^2)	26.5 ± 2.8	25.8 ± 2.4	28.0 ± 2.3	27.0 ± 2.0	26.5 ± 2.3
LVIDs (mm)	33.7 ± 4.8	34.0 ± 3.7	35.2 ± 4.2	34.7 ± 3.1	32.5 ± 4.8
LVIDs (mm/m^2)	16.8 ± 3.2	17.0 ± 2.4	17.7 ± 2.3	17.3 ± 1.5	16.2 ± 2.9
LV mass (g)	212.3 ± 25.3	212.4 ± 44.9	224.9 ± 37.6	205.7 ± 40.7	202.2 ± 35.8
LV mass (g/m^2)	106.3 ± 11.7	105.5 ± 21.7	112.8 ± 17.3	101.9 ± 18.8	99.7 ± 15.5
WS(kilodynes/cm^2)	54.8 ± 16.7	57.8 ± 19.9	56.7 ± 17.2	66.3 ± 15.3	54.8 ± 12.8
h/R	0.38 ± 0.08	0.39 ± 0.04	0.37 ± 0.05	0.36 ± 0.02	0.37 ± 0.05
FS	0.37 ± 0.08	0.35 ± 0.06	0.37 ± 0.05	0.36 ± 0.04	0.39 ± 0.7
Peak E (cm/sec)	63.6 ± 9.0	75.4 ± 10.3	68.5 ± 8.6	73.0 ± 12.8	69.4 ± 13.1
Peak A (cm/sec)	71.8 ± 17.9	68.7 ± 31.7	73.2 ± 17.8	75.6 ± 21.4	73.9 ± 23.2
E/A	0.94 ± 0.3	1.4 ± 1.0	0.98 ± 0.23	0.99 ± 0.14	1.0 ± 0.3

(Mean ± S.D.; VSTd, diastolic ventricular septal wall thickness; VSTs, systolic ventricular septal wall thickness; PWTd, diastolic posterior wall thickness; PWTs, systolic posterior wall thickness; LVIDd, left ventricular diastolic cavity diameter; LVIDs, left ventricular systolic cavity diameter; LV mass, left ventricular mass; WS, left ventricular end-systolic wall stress; h/R, relative wall thickness; FS, fractional shortening; Peak E, peak early diastolic flow velocity; Peak A, peak late diastolic flow velocity; E/A, peak early to late diastolic filling ratio; all comparisons $p>0.05$).

Table 6.2 Effects of sixteen weeks of resistance training on leg-press and bench-press maximal muscular strength

Variable	Baseline	Post 16 Weeks
Leg Press (kg)	RT: 284.8 ± 48.3 CT: 290.9 ± 58.8	366.9 ± 47.2# 289.7 ± 52.5*
Bench Press (kg)	RT: 59.3 ± 11.0 CT: 60.1 ± 9.0	68.9 ± 10.6# 61.0 ± 12.5*

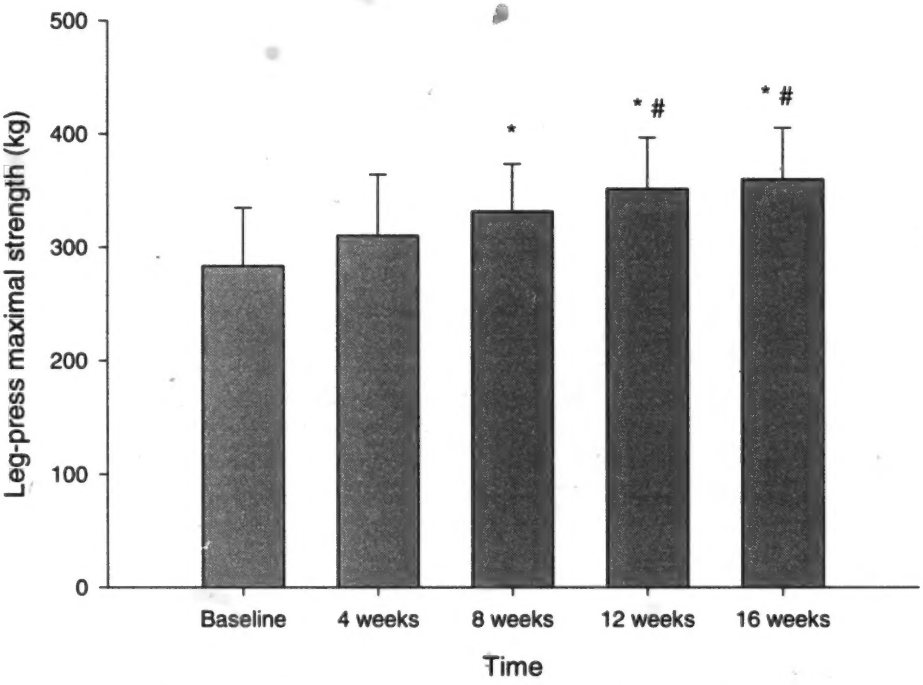
(Mean ± S.D.; RT, resistance training; CT, control group; *, $p<0.05$ vs RT; #, $p<0.05$ vs. Baseline)

Table 6.3 Main time effect for left ventricular dimensions, mass, systolic and diastolic function

Variable	Baseline	Post 16 weeks
LVIDs (mm)	33.4 ± 5.3	32.7 ± 4.7
(mm/m^2)	16.8 ± 2.6	16.5 ± 2.4
PWTd (mm)	10.3 ± 1.6	9.9 ± 1.0
(mm/m^2)	5.2 ± 0.9	5.0 ± 0.5
PWTs (mm)	16.0 ± 1.8	15.8 ± 1.7
(mm/m^2)	8.1 ± 1.2	8.0 ± 0.9
h/R	0.39 ± .07	0.39 ± .05
WS (kilodynes/cm^2)	56.3 ± 19.3	57.9 ± 14.3
FS	0.38 ± .07	0.37 ± .07
Peak E cm/sec	70.4 ± 12.8	68.7 ± 12.5
Peak A cm/sec	74.80 ± 16.0	73.30 ± 15.7
E/A	0.98 ± 0.30	0.97 ± 0.22

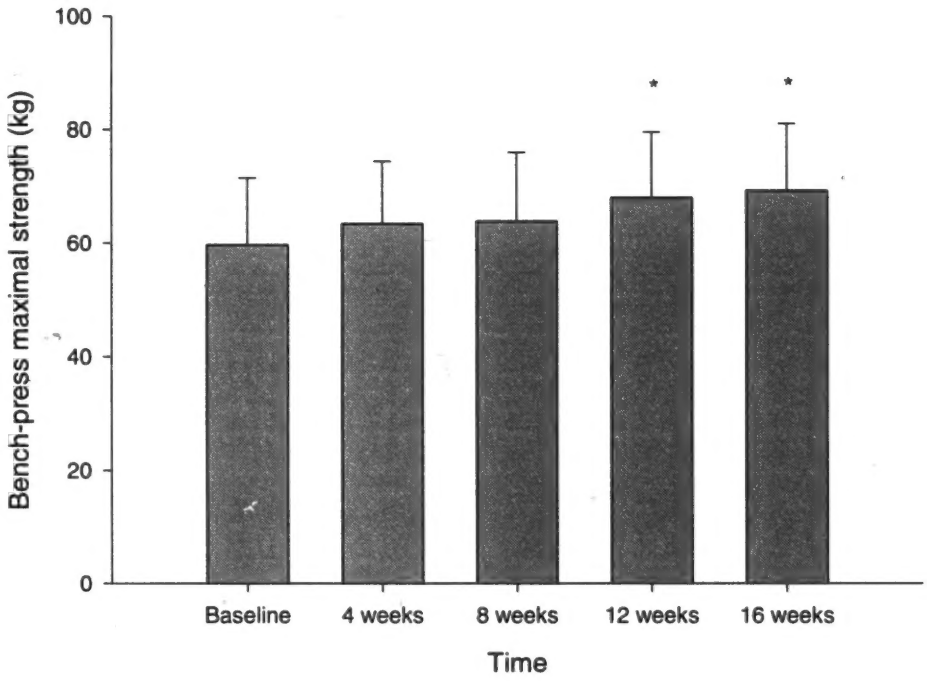
(Mean ± S.D.; LVIDs, left ventricular systolic internal dimension; PWTd, diastolic posterior wall thickness; PWTs, systolic posterior wall thickness; h/R, relative wall thickness; WS, left ventricular end-systolic wall stress; FS, fractional shortening; Peak E, peak early diastolic flow velocity; Peak A, peak late diastolic flow velocity; E/A, early to late diastolic filling ratio; all comparisons $p>0.05$).

Figure 6.1 Time course for the alteration in leg-press maximal muscular strength associated with 16 weeks of resistance training



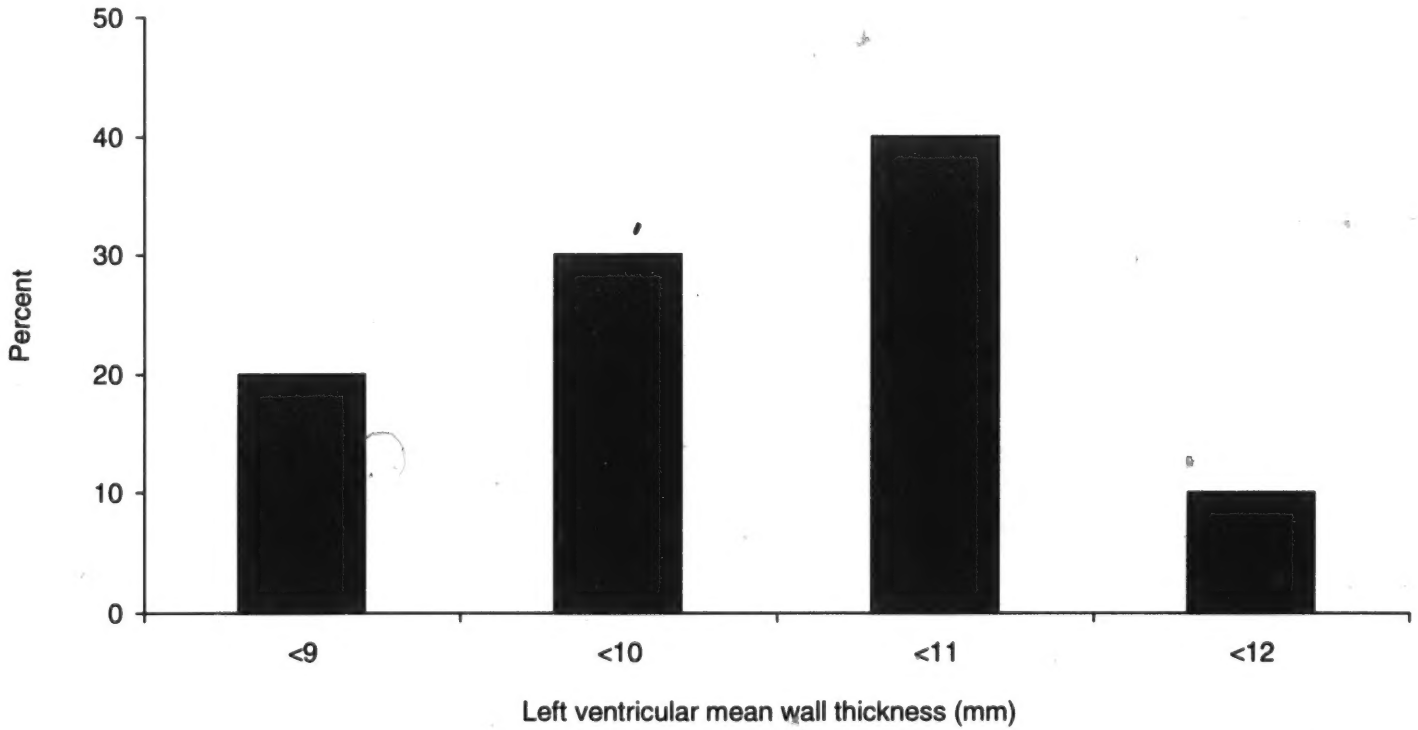
(mean +/- S.D.; *, $P < 0.05$ vs. baseline; #, $P < 0.05$ vs. week 4)

Figure 6.2 Time course for the alteration in bench-press maximal muscular strength associated with 16 weeks of resistance training



(mean +/- S.D.; *, P <0.05 vs. baseline)

Figure 6.3 Distribution of left ventricular mean wall thickness after 16 weeks of resistance exercise in healthy older males



General discussion

A widely held belief in sport cardiology has been that the transient abrupt increase in systolic pressure during RT was associated with a concomitant elevation in LV wall stress which may be a potent stimulus for LV hypertrophy (i.e., load-induced hypertrophy hypothesis, Colan, 1992). However, the major finding of chapter III was that resistance exercise was not associated with an acute alteration in LV end-systolic wall stress. The attenuated exercise wall stress was secondary to two independent mechanisms. Firstly, we found that the increased systolic pressure during resistance exercise was directly related to the elevated intra-thoracic pressure associated with performing a brief (~2 to 3 second) Valsalva maneuver. However, the pressure to which the heart was "exposed" (i.e. LV end-systolic transmural pressure) was not elevated above resting measures. Secondly, it was found that the LV alters its geometric configuration during heavy resistance exercise such that the tension per myocardial area was reduced, and as a consequence lowered LV wall stress. Therefore, acute heart-lung interactions combined with intrinsic changes in LV geometry may occur during resistance exercise to minimize the stress to which the heart was "exposed" and (in accordance with the load induced hypertrophy model) may reduce the primary stimulus for LV hypertrophy.

A limitation of our finding that resistance exercise was not associated with an acute alteration in LV wall stress was that it was limited to the concentric phase of the leg-press lift. Thus, the effects of a brief (~2 to 3 second) Valsalva maneuver on LV wall stress during eccentric leg-press resistance exercise is not

well known. However, Lentini et al., (1993) have demonstrated that the pressure to which the heart was "exposed" during eccentric leg-press exercise performed with a brief Valsalva maneuver was lower than that predicted by the intra-vascular pressure alone. Therefore, a brief (~2 to 3 second) Valsalva maneuver should not be discouraged during concentric or eccentric leg-press exercise (Lentini, et al., 1993).

The major finding of chapter IV and V was that short (< 5 years) or long-term (> 10 years) RT as performed by younger or middle-aged elite powerlifters was not associated with alterations in LV dimensions or estimated LV mass. Furthermore, 16 weeks of RT was not associated with changes in LV systolic or diastolic wall thickness, cavity size, relative wall thickness, estimated LV mass, LV wall stress, and LV systolic or diastolic function in healthy older males (Chapter VI). Together, these findings confirm and extend previous studies that found that RT was not associated with an alteration in LV morphology (Brown, et al., 1987; Elias, et al., 1991) and suggest that 0.3 to 18 years of RT may be a sufficient stimulus to increase maximal muscular strength but was insufficient of a stimulus to alter LV morphology.

Upper limits of LV wall thickening associated with resistance training

Pelliccia and associates (1993) assessed the upper limits of LV wall thickness in 100 younger (mean age: 26.5 years) resistance trained athletes and found that no athlete had a measured LV wall thickness above normal clinical limits (i.e., >12mm). Our finding that RT was not associated with a LV mean wall thickness above 12mm is consistent with that previously described. Together,

these findings suggest that if a junior or master RT athlete is found to have a LV mean wall thickness greater than 12mm then further investigations may be warranted (to rule out other forms of pressure overload hypertrophy) as this finding is an unlikely consequence of RT.

Conclusions

The major finding of this dissertation was that 0.3 to 18 years of RT was not associated with changes in LV wall thickness, relative wall thickness and estimated LV mass in younger, middle-aged or older males. Moreover, these findings appear to be related to the finding that this form of exercise was not associated with an acute or chronic increase in LV wall stress. Therefore, short or long-term RT appears to be a sufficient stimulus to increase maximal muscular strength, but is an insufficient stimulus to alter LV morphology. Lastly, RT can be described as a "safe" and effective intervention that can be performed by older adults to improve overall muscle strength without altering the size and systolic or diastolic function of the heart.

Future studies

RT has recently gained popularity as a safe and effective intervention to improve muscular strength for cardiac patients (Verill and Ribisil, 1996). Despite the benefits of this form of exercise, a recent investigation has shown that individuals with congestive heart failure (CHF) are capable of generating a relatively high systolic pressure (i.e., 189mmHg) when performing sub-maximal resistance exercise (McKelvie, 1995). Therefore, it may be possible that the increased systolic pressure may lead to an elevation in LV wall stress in individuals with a compromised LV. At the present time, the acute or chronic effects of the heightened pressure load on LV morphology, systolic and diastolic function in individuals with CHF are not well studied. However, before this form of training can be classified as "safe" for individuals with CHF further studies are required to ensure that this form of training does not alter the size, shape or function of their heart.

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